A CLINICAL STUDY

OF

OCULAR SIGNS AND SYMPTOMS ASSOCIATED WITH INTRACRANIAL TUMORS

DISSERTATION

SUBMITTED TO

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In partial fulfillment of the regulations for the

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CERTIFICATE

I hereby certify that this dissertation entitled **" A CLINICAL STUDY OF OCULAR SIGNS AND SYMPTOMS ASSOCIATED WITH INTRACRANIAL TUMORS"** is a bonafide work by **Dr. K. Divya,** during the period from February 2004 to March 2006 in **Coimbatore Medical College,** Coimbatore under my guidance and supervision. The conclusions reached in this study are her own. I have great pleasure in forwarding it to the Tamilnadu Dr. MGR Medical University.

Place : Coimbatore

Date :

Head of the Institution **Head of the Department**

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INTRODUCTION

INTRODUCTION

Developmentally, the visual system is an outgrowth from the central nervous system. Morphologically and functionally, the visual system, as a whole can be called as little brain[46.]

Tumors of the brain, its meningeal coverings, and spinal cord rank second only to stroke as the most common neurologic cause of death.

Primary brain tumors account for 20% of all malignant tumors in children, second only to leukemia and about 2% of all cancers in adults.

More than 50% of patients with intracranial tumors first produce ocular signs / symptoms / both. They are first seen by an ophthalmologist at an early stage, where there is greatest chance for optimum treatment of these lesions.

Role of ophthalmologist [51] in the clinical management of neurologic tumor may involve:

1. Recognition of signs and symptoms suggesting intracranial neoplasm.
2. Use of appropriate diagnostic tests like CT / MRI, to distinguish tumor from other causes of progressive neuro ophthalmologic dysfunction.
3. Urgent referral of patients to neurologist for management .
4. Appreciation of prognosis and complications of management.
5. Awareness of treatment controversies and alternatives.

Brain Tumors

Incidence

In persons younger than 20 years of age, CNS tumors represent 20% of malignancies. Two thirds of tumors in this age group are in the posterior fossa.[51]

Common tumors of childhood and adolescence are astrocytomas of cerebellum, brainstem and optic nerve, pinealomas and craniopharyngiomas. Common tumors of midlife (20-60yrs) include meningiomas, gliomas of cerebral hemispheres and pituitary tumors. Malignant astrocytomas and metastases represent bulk of tumors in late life. With exception of meningiomas and acoustic neuromas, most intracranial tumors are slightly more common in men.[51]

Frequency Distribution Of Brain Tumors [51]:

Adults	Children
Glioma (40-67%)	Medulloblastoma (20-25%)
Meningioma (9-27%)	Cerebellar astrocytoma (12-18%)
Pituitary adenoma (15%)	Brainstem glioma (6-15%)
	Supratentorial astrocytoma (14-32%)

An increase in incidence of gliomas in elderly has been reported in the recent years. This may be real or the result of more frequent use of modern imaging techniques.[52]

Classification of Brain Tumors [51]

Primary Tumors

1. Tumors of glial origin
2. Tumors of neuronal origin
3. Tumors of neural crest origin
4. Tumors of connective tissue origin
5. Tumors of lymphoreticular origin
6. Tumors of glands
7. Tumors of embryonal cell rests
8. Tumors of Blood vessels
9. Tumors of Skull

Metastatic Tumors

1. Melanomas (50-80%)
2. Lung (20-30%)
3. Breast (20%)
4. Kidney (8-20%)
5. Colorectal (1-5%)
6. Ovarian (1%)

(Modified from Escourolle R, Poirier J; manual of neuropathology. II Edn. Philadelphia, WB Saunders (1978))

Clinical Manifestations

The symptoms and signs caused by Brain tumors are determined by their size, location, invasiveness and rate of growth[49].

Patients with Brain tumors usually have one or more of following groups of symptoms.

1. Headache with or without evidence of increased intracranial pressure.
2. Progressive generalized decline in cognitive abilities or in specific neurologic functions such as speech, gait or vision
3. Adult onset seizures
4. Focal neurologic symptoms and signs.

Topical Diagnosis of Tumors

Clinical features of intracranial tumors can be categorized as

1. Non localizing signs and symptoms
2. False localizing signs and symptoms
3. Truly localizing signs and symptoms

Non Localizing Signs and Symptoms

These are independent of site tumor and primarily indicate an increase in intracranial pressure.

Headache

- Headache is among the initial symptom in 20% of patients with brain tumors.
- It appears in 70% of patients sometime during course of their illness.
- Usually headache is of mild to moderate severity, worse on the side of lesion, worse on bending down and associated with nausea and vomiting .
- Headache is more common in tumors below tentorium.
- Pain arising from superior surface of tentorium cerebelli is referred to forehead and periorbital regions by branches of trigeminal nerve.
- Pain arising from inferior surface of tentorium cerebelli and posterior fossa is referred to the throat and ear by IX and X cranial nerves and to the occipital and nuchal regions by the first three cervical spinal nerves.
- Tumors around optic chiasm, particularly pituitary adenomas cause characteristic “bursting” headaches that are felt at the bifrontal areas.

Mechanism of Headache

1. Sudden changes in intracranial pressure (whether increase / decrease)
2. Traction upon or displacement of pain sensitive intracranial structures.
3. Acute hemorrhage into tumor
4. Venous sinus thrombosis
5. Acute obstructive hydrocephalus
6. Contiguous involvement of bone / meninges
7. Toxic effects of various therapies.

Detailed neurologic and ophthalmic Examination is indicated in :

1. Intense / Prolonged / Incapacitating headache.
2. Changes in frequency / pattern / quality of headache.
3. Recent onset of increased vomiting with headache.
4. Associated with seizures or other focal neurological abnormalities.
5. Headache that awakens individual from sleep.

Vomiting

Vomiting occurs in 70% of patients at sometime during the illness. Projectile nature, especially if occurring without relationship to intake of food, and unassociated with nausea, should arouse suspicion of increased intracranial pressure.

Seizures

Tumor is a major etiologic consideration in a patient with adult onset seizure disorder, particularly in patients between fourth and sixth decades of life.

Supratentorial tumors induce seizures more frequently, than infratentorial ones. Supratentorial tumors that are located in the temporal lobe and near the motor and sensory cortexes are more epileptogenic than those situated in the anterior fontal lobe, parachiasmatic region or occipital lobe.

There is no precise correlation between seizure pattern and tumor location – But usually a seizure associated with forced turning of head and eyes (versive movement) indicates a tumor contralateral to the direction of head and eye movement.

The more slowly the tumor grows, the higher the risk of a seizure.

Papilledema

- Papilledema is the classic and most important sign of increased intracranial pressure.
- Defined as passive non-inflammatory swelling of the optic disc caused by raised intracranial pressure.
- Papilledema is uncommon in infants and children less than 2 years due to non fusion of cranial sutures, which allows room for enlargement.
- Papilledema occurs more frequently between the ages of 2 and 10 years due to the high frequency of infratentorial tumors in this age group.
- Papilledema is rarely encountered in elderly patients, because of the low incidence of brain tumors in this age group.
- Infratentorial tumors from cerebellum and fourth ventricle tend to increase intracranial pressure early by obstructing flow of cerebrospinal fluid through aqueduct of sylvius.
- Among supratentorial tumors, III ventricle tumors cause papilledema frequently.
- Overall 60 percent of patients with cerebral tumors have papilledema sometimes during course of their illness.
- Three regions of brain, where tumors usually develop, without causing papilledema, are the pons, pituitary and central white matter of the cerebral hemispheres.[43]

Mechanism of Increased ICT With Intracranial Tumors.

1. Increase in total amount of intracranial tissue.
2. Increase in intracranial volume due to cerebral edema (focal / diffuse)
3. Blockage of CSF causing hydrocephalus(obstructive / communicating)

4. Decreased absorption of CSF due to compromised cerebral venous outflow
5. Increased CSF production.[45]

Symptoms

1. Transient obscurations of vision lasting 5 – 30 seconds, that occur at irregular intervals, often precipitated by changes in posture.
2. In long standing cases, if optic atrophy sets in, visual acuity falls with progressive constriction of fields.

Signs

1. Hyperemic disc with blurred margins and filled up cup.
2. Loss of spontaneous venous pulsation.
3. Venous engorgement and peripapillary flame shaped hemorrhages.
4. Fundus fluorescein angiography initially shows dilated disc capillaries followed by increased hyperfluorescence which extends beyond the margin.
5. Fields show enlargement of the blind spot.

False localizing signs and symptoms

- Defined as a sign which potentially causes confusion in diagnosis by suggesting an abnormality at a distance, away from the actual site of lesion.

Mechanism :

1. General compression of a nerve having a long course
2. Meningitis
3. Edema and gliosis
4. Metastatic infiltration
5. Hydrocephalus
6. Gross brain displacement with shift of Sagittal plane

VI Cranial nerve is affected more commonly [29,30] as it gets compressed between pons and basilar artery / stretched along sharp edge of petrous bone.

Downward displacement of the temporal lobes through the tentorium due to a large hemisphere mass may cause temporal coning. This may stretch the III / VI Cranial nerve or cause pressure on the contralateral cerebral peduncle resulting in ipsilateral upper motor neuron signs.

Dementia, visual field defects, hallucinations, cerebellar signs, pyramidal and extrapyramidal signs, nystagmus, etc., can manifest as false localizing sign at one time or other.

Truly localizing signs and symptoms

Tumors of frontal lobe

Gliomas (astrocytomas, oligodendrogliomas and glioblastoma multiforme) are the most common frontal lobe tumors. Withdrawn behaviour, apathy, senseless joking, irritability, lack of judgement etc, constitute the major cognitive symptoms.

Motor signs include forced grasping, groping and spasticity in the contralateral limbs. Aphasia, focal motor and jacksonian seizures may also occur.

Slowly progressive mono ocular visual loss with central scotoma is the significant clinical ocular manifestation, caused by pressure on the underlying optic nerve by a large frontal lobe tumor.

Damage to frontal gaze centre results in defective saccades to opposite side. Conjugate horizontal deviation of eyes with head turning away from side of tumour, is observed as part of the “adversive Seizure”.

Foster Kennedy Syndrome: Ipsilateral optic atrophy, with contralateral papilledema, is a sign of great localizing value.[50]

Tumors of Temporal Lobe

About 10% of all intracranial and 20% of all supratentorial tumors involve this lobe. More common tumors of the temporal lobe are gliomas, meningiomas and metastatic tumors.

Psychomotor seizures constitute the most outstanding and interesting symptom complex of temporal lobe tumors.

Olfactory or gustatory hallucinations, deja Vu (feeling of familiarity) and jamais Vu (feeling of strangeness) are a frequent occurrence.

Visual hallucinations of formed images, micropsia, macropsia and telopsia may be experienced.

Visual field defects are the most conspicuous. Involvement of the Meyer's loop forms the anatomical substrate for field defects in patients with temporal lobe tumors.

Most common type of defect is an incongruous homonymous hemianopia, with sloping margins, often denser in the superior quadrant and in the eye ipsilateral to the tumor.

Field defect can begin as a contralateral upper homonymous superior quadrantanopia called "pie in the sky" defect.[31]

Lesions of dominant temporal lobes produce wernicke's aphasia, and impairment of tests of verbal material presented through the auditory sense.

In the non dominant temporal lobe lesions, there will be impairment of tests of visually presented non verbal material, agnosia for sounds and inability to judge spatial relationships.

Tumors of Parietal lobe

Gliomas, particularly glioblastoma multiforme, convexity and parasagittal meningiomas, vascular tumors, ependymomas and metastatic tumors are the most common tumors.

Field defects usually start as inferior homonymous quadrantanopia or "pie on floor" defect. Deep seated parietal lobe lesions produce a positive OKN sign. When the drum is rotated towards the "diseased" hemisphere, the OKN response will be impaired or asymmetric. Visual inattention is also a characteristic feature.

Cogan's sign of spasticity of conjugate gaze.

In patients with parietal lobe lesions, forced lid closure leads to conjugate deviation of eyes to the side opposite the lesion.

Gerstmann Syndrome:

Finger agnosia, right left disorientation, acalculia and agraphia constitute a lesion in the dominant lobe.

Lesions of non dominant lobe result in neglect of one half of visual field, and dressing apraxia. Involvement of sensory cortex (post central gyrus) produces cortical sensory loss on opposite side which manifests as loss of touch, pain and position sense.

Tumors of Occipital lobe

Neurologically these are silent zones. Visual field defects which are largely congruous are the main features. Complete hemianopia with macular sparing is the typical presentation.

Cogan's rule

Asymmetric optokinetic nystagmus response in a patient with occipital lobe lesion indicates the lesion is neoplastic in nature, rather than vascular.

Visual hallucinations are unformed and may precede the development of hemianopia.

Riddoch phenomenon :

A dissociation of visual perception where in motion is perceived in a field, that is otherwise blind to form.

Visual agnosia (failure to recognize objects in absence of visual impairment), color agnosia, prosopagnosia (inability to recognize familiar faces) and palinopsia (persistence of visual image after the stimulus is removed from the visual field) are other manifestations.

Tumors in the parasellar area:

These tumors mainly include sphenoid ridge meningiomas and cavernous sinus tumors. The sphenoid ridge tumors can be divided into

1. Those arising from outer 1/3 rd of sphenoid ridge.
2. Those arising from middle 1/3rd of sphenoid ridge.
3. Those arising from inner 1/3rd of sphenoid ridge.

They produce ocular symptoms if they expand in a medial direction and involve the optic nerve and superior orbital fissure. Mono ocular visual loss and proptosis are by far the most common ophthalmic findings of sphenoid ridge meningiomas. Characteristic feature is unilateral exophthalmos with variable paresis of III, IV and VI cranial nerves and occasional hypoaesthesia of ipsilateral cornea.

Tumors in Sella and Suprasellar region

Most common tumors[32] in this region include pituitary adenomas (50-55%) Craniopharyngiomas (20-25%), meningioma of tuberculum sella (10%), optic chiasmal and hypothalamic gliomas (7%)

Pituitary adenoma :

Pituitary adenoma occurs most frequently between fourth and sixth decades of life. Classical field defect is bitemporal hemianopia or quadrantanopia, that respects the vertical midline.

The field defects produced depend upon the shape and general configuration of pitiutary fossa and relationship of chiasma to it (prefixed / postfixed).

If the chiasm is central, the field defect starts as a superior temporal quadrantanopia and then progresses to inferior quadrant, since the tumor causes compression from below and affects the lower nasal fibres first.

About 50% of patients with chromophobe adenomas have endocrine disturbances like delayed body growth (pituitary dwarfism), adiposogenital dystrophy (Frohlich's syndrome), sexual disturbances (amenorrhea, loss of libido) weight gain etc.,

Eosinophilic adenomas cause overproduction of GH and cause acromegaly / gigantism. Basophilic adenomas secrete excessive ACTH and produce cushing's syndrome.

Sudden decline in vision with extra ocular muscle palsy and altered consciousness indicates a pituitary apoplexy resulting from hemorrhage or infarction into a pituitary adenoma.[42]

Syndrome of Distal Optic Nerve

(Anterior chiasmal syndrome)

Compression of intracranial part of optic nerve leads to central or arcuate scotomas, peripheral constriction or hemianopic field loss. Decussating fibres from nasal half of retina loop anteriorly into the contralateral optic nerve before they go on to the optic tract – Damage to this area (anterior knee of wilbrand) produces a characteristic field defect – Junctional scotoma of traquair.

Craniopharyngioma

Slow growing tumor arising from vestigial remnants of Rathke's pouch along the pituitary stalk[44].

These compress the chiasm from above. Visual field defect starts in the inferior quadrant and proceeds upwards.

Associated features in children include, dwarfism, delayed sexual development and obesity due to interference with hypothalamic function[35].

Tumors Of Cerebello Pontine Angle

These include schwannoma of the VIII nerve, meningioma, ependymoma, cholesteatoma and osteomas.

Tinnitus and hearing loss are early symptoms due to involvement of cochlear division of VIII cranial nerve.

Earliest sign is diminished corneal sensation.

Other features include paresis / paralysis of fifth, sixth and seventh cranial nerves, inco-ordination, ataxia and cerebellar signs.

Tumors Involving Superior Orbital Fissure And Cavernous Sinus

Tumors in this region can manifest as :

Spheno Cavernous Syndrome

Constitutes paralysis / paresis of more than one ocular motor nerve, with I and II divisions of trigeminal nerve, Horner's syndrome and visual loss

III CN paresis associated with small pupil

Can occur due to involvement of both III CN and the sympathetic pathway in the cavernous sinus where they are closely related.

VI CN Paresis with ipsilateral Horner's Syndrome

Can result because the sympathetic fibres join briefly with VI CN, before they merge with the ophthalmic division of V cranial nerve.

Primary oculomotor nerve synkinesis

(Primary aberrant regeneration)

It is a condition in which misdirected innervation of extra ocular muscles normally innervated by III cranial nerve occurs without any previous history of acute III CN paresis.

Commonly occurs with slow growing lesions of cavernous sinus eg. Meningiomas or aneurysms.

Tumors of pineal gland

Atypical teratomas are the most common among these tumors, with maximum incidence between first and second decades.

Precocious puberty may be the presenting feature in some. Characteristic ophthalmologic features include supranuclear vertical gaze palsy with pupillary light near dissociation and convergence retraction nystagmus (Parinaud's syndrome)

Metastasis

Multiple lesions in brain are characteristic of secondaries. Breast and lung carcinomas are the leading primary tumors. Signs depend on the site of involvement.

INVESTIGATIONS

Preliminary investigations

These are done to rule out any infection / diabetes / hypertension which need to be treated / controlled before surgery in the patient.

The blood count and peripheral blood smear will also be useful in cases of suspected primary malignancy elsewhere giving rise to secondary matastasis in brain.

Special investigations.

Neuro imaging techniques complement the patient history and physical examination.

Computed Tomography

Patient is moved through a circular structure, with an x-ray emitter located across from a detector. The emitter is rotated 180^0 and the computer analyses the density of target tissues from attenuation values, reconstructing an image.

CT is an excellent method for evaluating bony destruction in orbit, sella and clivus [40].

CT Scan

Advantages

1. Images can be obtained in less time
2. Superior in detecting bone, calcification and acute intracranial haemorrhage
3. Wider availability
4. Costs less

Disadvantages

1. Relatively poor soft tissue contrast
2. Artefacts from bone and metallic objects
3. Lack of direct sagittal scanning capability
4. Difficulty in positioning patients to perform a coronal Scan

Magnetic Resonance Imaging

Neuro imaging study of choice for demonstration of intracranial soft tissue anatomy and pathology.

The target tissue is subjected to a strong magnetic pulse, and the energy emitted from the recovering tissue is converted to an image.

Views can be obtained directly in any plane allowing excellent localization and three dimensional reconstruction of images.

Advantages

1. Multiplanar capability
2. Increased soft tissue contrast
3. Preferred for demonstration of sellar and suprasellar, masses, posterior fossa lesions, vascular malformations and aneurysms

Disadvantages

1. Requires a scanning time of several minutes per sequence
2. Difficult in claustrophobic patients
3. Contra indicated in patients with cardiac pace makers, metallic implants. etc.,
4. More expensive

Treatment

Management of an intracranial tumor is primarily by the neurosurgeon. Surgery is indicated for definitive diagnosis and for possible debulking of most primary brain tumors. The surgery can either be

1. Total excision
2. Subtotal excision and biopsy.

Biopsied material is sent for histopathological examination, based on which the patient is subjected to further surgery or radiotherapy.

The role of the ophthalmologist is to assist the neurosurgeon in decisions regarding the time of surgical intervention so as to prevent further progression of the lesion.

AIM

AIM OF THE STUDY

1. To study the value of ocular signs in predicting the presence of an intracranial tumour.
2. To correlate the ocular symptoms and signs with the location of the brain tumor.
3. To study the changes in ocular signs with treatment.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

No other portion of the visual pathway offers a better opportunity for exact correlation between anatomy and function than the chiasm [47].

Chromophobe adenomas are the commonest type of pituitary tumors, occurring typically in adults above the age of 30[48]. Mechanical effects of their growth vary considerably. If the fossa is shallow with anterior wall poorly differentiated, expansion in all directions is easy. If the fossa is deep and its lips well formed, the tumor tends to be retained and lateral growth against the cavernous sinuses is encouraged [48].

Approximate vertical distance of 10 mm separates chiasm from dorsum sella and pituitary fossa. Sellar region lesions must already be spreading to suprasellar region, before patient can notice visual field defects[1,2].

Thomas R et al[3] studied the prevalence and pattern of visual field loss in non functioning pituitary adenomas and relationship between the tumor size and severity of field defects. Ninety three patients with histologically confirmed pituitary adenomas, non functional on hormonal assessment underwent a complete ophthalmic evaluation and automated perimetry using the HFA – 30-2 programme. Eighty eight of ninety three (94.6%) patients had a field defect. Typical field defects were seen in 69 (74.2%) patients and atypical field defects in 19 (20.4%). All 31 patients with a tumor size greater than 20 cc had field defects. Severity of field defects increased with tumor volume. (chi square test for trends significant p = 0.0096) A severe visual field loss involving at least 3 quadrants in one or both eyes was the most common.

Harper A et al[4,34], conducted a prospective survey of 29 patients with pituitary macroadenomas who presented to the neurosurgical unit at St.

Vincent's Hospital, Melbourne. All patients had visual field defects detected on perimetry and the majority were asymmetrical. Bi-temporal defects were most common but field defects ranged from monocular defects to generalized constriction. Four patients (13.8%) did not report visual symptoms, and of those who had symptoms, blurred vision was the most common complaint. Ninety six percent of eyes had field loss, 56% had decreased color vision, 46% had decreased acuity, 31% had disc pallor and 2% had an ophthalmoplegia.

They concluded that perimetry is the most sensitive method of identifying compression, and automated static threshold perimetry appears to show early field defects better than manual kinetic perimetry.

Measurement of Retinal nerve fibre layer thickness using scanning laser polarimetry also provides useful information in the diagnosis of chiasmal lesions such as pituitary adenomas[5].

Sato M et al[6], examined the usefulness of the Bagolini striated glasses test to identify patients with a lesion of the optic chaism. Nine out of twelve patients (75%) showed conflicting results with the Bagolini striated Glasses test between monocular and binocular conditions. With monocular testing essentially normal stripes were observed in each eye. In binocular testing, stripes which were projected upon nasal retina were suppressed bilaterally (bitemporal hemianopia - "mountain pattern") or monocularly (monocular temporal heminanopia).

The sensitivity of transient pattern – reversal visual evoked potentials in the detection of early compressive lesions of the chiasm is controversial in the literature[7]. However there have been claims that, the technique is capable of detecting an abnormality in the absence of any demonstrable visual field loss, when large field and check sizes were used.

Porciatti v et al[8], studied the function of contrast sensitivity in patients with pituitary adenomas with normal visual acuity and visual fields. CS was evaluated in both hemifields of 28 patients, using two different stimuli: a coarse (0.3c/deg) dynamic (10HZ) grating and finer (2c/deg) static grating. CS losses were more frequent for 2 c/deg static stimuli. CS evaluation may provide a simple and effective tool for early detection and monitoring of visual dysfunction in patients with pituitary adenoma.

The visual loss associated with compression of the optic chiasm by pituitary tumors may be transient or permanent, possibly related to the extent of irreversible retrograde degeneration of the retinal ganglion cells. The pattern ERG N95 component is thought to rise in relation to retinal ganglion cell function and hence may be a potential prognostic indicator for visual function following decompressive surgery [9].

Kerrison et al[10], studied the stages of improvement in visual fields after pituitary tumor resection using static threshold perimetry. The pattern of recovery suggested at least three phases of improvement. The early fast phase (surgery to 1 week) of improvement may lead to normalization of visual field in some individuals. The early slow phase (1 month to 4 months) is the period of most notable improvement. A late phase (6 months to 3 years) of mild improvement does not appear significant overall but may be marked in some individuals.

Chen z et al,[11] evaluated the clinical application of Humphrey three zone screening in detecting the visual field of the patients with pituitary macroadenomas. 99.2% of the 128 patients and 87.1% of their eyes had visual field defect 86.7% of the patients had the visual field defect mainly located at the temporal side and showed or tended to have a medial vertical limit. Humphrey three zone screening has a very high sensitivity and specificity in

detecting visual field and it is simple, fast and practical for detecting the visual field of the patients with pituitary macroadenoma. It plays an important role in implying diagnosis and avoiding misdiagnosis of pituitary macroadenomas.

Rare Presentations of a pituitary adenoma include visual hallucinations[12] and amaurosis fugax [13].

Lee Ag et al[14] report a case of progressive visual loss after trans sphenoidal resection of pituitary adenoma. A 63 year old man, who underwent uneventful trans–sphenoidal resection of a pituitary adenoma with fat packing complained postoperatively of binocular visual loss. Neuro imaging showed a suprasellar pneumatocele compressing the optic chiasm and a communication between the sphenoid sinus and sella. Suprasellar pneumatocele probably forms through a ball valve mechanism that results from incomplete packing of the sellar floor. This case highlights the need for effective sphenoid sinus packing and for ophthalmic monitoring after trans sphenoidal surgery.

Although the diagnosis of pituitary adenoma is usually inferred from the results of neuro imaging, lesions other than pituitary adenomas can have an appearance that suggests an adenoma.

Miller et al[15] did a retrospective case controlled analysis of medical record data to determine whether there are clinical findings that suggest a lesion producing a chiasmal syndrome is something other than a pituitary adenoma. The search revealed 149 patients who met the inclusion criteria, including 90 patients with pituitary adenomas and 59 patients with other lesions. Variables that were highly suggestive of an etiology other than pituitary adenoma included symptomatic visual loss, younger age unilateral optic disc pallor, a relative afferent pupillary defect and an absolute or a complete visual field defect or one that was greater inferiorly than superiorly.

Pituitary adenoma is an uncommon intracranial tumor in children. However ophthalmologists should be aware that pituitary adenomas may occur in children [16,39] and that these tumors when present in pubertal period may be more likely to exhibit extrasellar extension or invasiveness.

Craniopharyngiomas is the most convenient term to describe the group of congenital tumors and cysts which arise from remnants of the epithelial tract from which Rathke's pharyngeal pouch, the anterior lobe of pituitary and hypophyseal duct are formed. As a rule, symptoms appear in childhood or adolescence (average 10 to 25 years). Frequently however, it remains small and stationary for many years and may not give rise to symptoms until adult life[36] (64 years, Rintelen and Leue N Berger, 1957; 40 years, Grossi and Bardelli, 1965)

Crompton JL et al[17] did a retrospective case review to assess the clinical presentation and long term visual outcome in a series of patients with craniopharyngioma. Thirty six patients were reviewed comprising 19 female and 17 male patients. The age range was 2-77 years with a bimodal distribution of 17 children (mean age 10 yrs) and 19 adults (mean age 47 years). The results were as follows.

Blurred vision	-	64%
Headache	-	53%
Average duration of systemic symptoms	-	45 weeks
Average duration of visual symptoms	-	10 weeks

Children are more likely to present with systemic symptoms than adults. Visual field pleomorphism is a feature of craniopharyngioma and occurred in one third of the patients. Although MRI is the recommended means of follow

up, regular neuro ophthalmic review is useful in the early detection of anterior visual pathway compression by recurrent tumor.

Craniopharyngioma[18], presenting as a case of unilateral central visual loss has also been reported.

Symptomatic Rathke's cleft cysts can also manifest as visual disturbance[19,38]. Every effort to distinguish Rathke's cleft cysts from craniopharyngiomas should be made preoperatively, for the former require only limited surgical intervention and radiotherapy is not necessary.

Lessells et al[20], performed a masked, controlled and quantitative measurement of the optic disc cup to determine if compressive lesions of the afferent visual pathway were associated with increased cupping. The ratio of cup: disc area of 29 patients with intracranial lesions impinging on the optic nerves and the chiasm (14 with pituitary adenomas, 7 with meningiomas, 6 with craniopharyngiomas and 2 with aneurysms) was compared with those of 20 age matched control subjects. The median ratio of cup: disc area was 0.37 for all eyes with visual compromise and 0.10 for control eyes, which was statistically significant ($p=0.0001$). This shows that several types of compressive lesions of the anterior visual pathway can be associated with increased cupping of the optic disc in the absence of increased intra ocular pressure.

Computer assisted interpretation of resolution visual fields can provide considerable support to the final diagnostic decision in patients with lesions of the visual pathways [21].

Acoustic neuromas comprise 8% of all primary intracranial neoplasms. Harner SG et al[22], reviewed 100 cases of pathologically confirmed acoustic neurinomas and compared tumor size, based on observations at operation with

findings on history and physical examination. Of the 100 patients, 36 had decreased corneal reflex, 36 had nystagmus, and 8 had papilledema. 92% of patients with nystagmus had brainstem compression from tumor. A positive correlation was found between tumor size and presence of signs and symptoms. Tumors causing nystagmus were atleast 2 cm in greatest dimension, those causing diminished corneal sensation were at least 2.5 cm and those causing subjective symptoms and papilledema were at least 4 and 4.5cm respectively.

Magnusson et al (1988)[23] reported that oculomotor dysfunction, defined as disturbed pursuit eye movements and / or gaze nystagmus was frequently found in patients with acoustic neurinomas larger than 2 cm (77%). A combined view of the oculomotor and caloric test results offers a possibility to obtain a rough estimate of tumor size as well as to distinguish acoustic neuromas from other types of tumors in the cerebello pontine angle.

Electronystagmography [24,33] may reveal damage to the vestibular brain-stem and archicerebellar structures, the ENG signs of which very often precede the clinical signs of compression. Vestibular and oculomotor instrumental investigations yield valuable clues for early diagnosis of tumor transition from the otological to the neurosurgical stage.

Hayreh SS et al[25] simulated progressively growing intracranial space occupying lesions in 32 rhesus monkeys by balloons introduced into the subarachnoid space of the temporal region. Optic disc edema first appeared in the lower pole, then the upper pole, then the nasal part and last the temporal part of the disc. The atrophic part of the disc did not develop edema. The studies indicated that swelling of the optic disc is the first sign of raised intracranial pressure and is due to swelling of the nerve fibres in the optic disc, the various associated vascular changes being secondary.

TR Hedges [26] et al studied 32 patients, with either crowded optic nerves or mild papilledema, with optical coherence tomography to determine the degree to which OCT can distinguish differences in the retinal nerve fibre layer thickness between eyes with mild papilledema, pseudopapilledema and normal disc. Circular OCT scans using a diameter of 3.38 mm surrounding the optic disc were performed in each eye of patients and subjects. Fundus photographs were analysed by two observers who diagnosed crowding or papilledema and graded amounts of swelling. Optic disc swelling was graded using a modification of the Frisen criteria.

Normal - diameter of optic cup between 10 and 50% of the overall optic disc diameter, no blurring of optic disc borders for 360^0 , and visibility of normal nerve fibres in all four quadrants.

Stage o ;- Blurring of the superior and inferior region of the optic disc margin with visible nasal and temporal optic disc margins.

Stage 1 ;- Additional nasal blurring with or without haemorrhage and a peripapillary halo.

Stage 2 ;- Additional blurring nasally and temporally with or without haemorrhage and with or without retino choroidal folds.

Stage 3 ;- (moderate Papilledema) Blurring of all four quadrants of disc margin with total obscuration of major vessel segments by swollen axons.

Stage 4 ;- Obvious, well established papilledema with haemorrhages and exudates.

OCT demonstrated measurable differences in RNFL thickness between normal subjects and patients with papilledema or pseudopapilledema. However OCT does not appear to differentiate between those individuals with

congenitally crowded optic nerves and those with mild papilledema caused by raised intracranial pressure.

Sixth nerve palsy is considered as a major false localizing sign and spontaneous recovery of a sixth nerve palsy is thought to rule out a neoplastic origin.

Lessell S et al[27] reviewed cases of VI nerve palsy that improved without treatment but that ultimately proved to be caused by a tumor at the base of the skull. Thus spontaneous recovery of a VI nerve palsy can occur in the presence of extramedullary compression by a tumor in the base of the brain.

Possible mechanisms for recovery include remyelination, axonal regeneration, relief of transient compression (resorption of haemorrhage), restoration of impaired blood flow, slippage of a nerve previously stretched over the tumor, or the immune responses to the tumor.

The clinical dictum that pupil sparing in oculomotor nerve palsy predicts an extra axial ischemic lesion while pupil involvement predicts an extra axial compressive lesion also has important exceptions [28,41].

Fast spin echo magnetic resonance imaging appears useful for objectively evaluating the optic nerve and surrounding subarachnoid space in patients with papilledema and optic atrophy [37].

MATERIALS AND METHODS

MATERIALS AND METHODS

The study was conducted at the Coimbatore Medical College Hospital, Coimbatore. The period of study was from February 2004 to March 2006. Out of the sixty five patients screened, fifty one patients were included in this clinical study based on the following guidelines.

Inclusion Criteria

The patients who had typical CT scan abnormalities suggesting a brain tumor, were evaluated and followed up.

Those patients who manifested with ocular signs suggestive of increased intracranial tension / compressive lesion in the brain were also evaluated with CT / MRI and included in the study.

Exclusion Criteria

Patients who had typical symptoms and signs suggestive of a space occupying lesion, but were found to have normal CT scans on investigation were excluded from the study.

Out of the intracranial space occupying lesions, lesions other than tumors. (eg. Abscess, tuberculoma, hematoma etc.,) were not included in this clinical study.

Neurologic Evaluation

A detailed history was taken about the complaints with which the patient first consulted a physician. Special emphasis was laid on.

1. Headache - Site, type, association with vomiting
2. Motor Symptoms - Weakness / paralysis or any seizures

3. Mental changes	-	Any irritational behavior of sudden onset or depression
4. Endocrine abnormalities	-	Growth retardation, menstrual disturbances, Galactorrhoea, decreased libido, delayed secondary sexual characters, polydipsia, polyuria, increased sweating,
5. Gait disorder		
6. Cranial nerve abnormalities	-	History relating to abnormal cranial nerve function was elicited by direct questioning, as many patients did not attribute much importance to the same and hence did not volunteer the information on their own.

A systematic central nervous system examination was carried out in all patients under the following groups:

- Higher mental functions
- Cranial nerve examination
- Motor and sensory system
- Examination for cerebellar / meningeal signs

Ophthalmological Examination

The history regarding any visual disturbances, occurrence of diplopia, protrusion of eyeball (proptosis), drooping of upper eyelid (ptosis) and limitation of ocular movement was noted.

Ophthalmic examination included a detailed evaluation of both eyes. Facial asymmetry, position of eyeball, eyelids and ocular movements were noted.

Pupils were examined for their size, shape, reaction to light – both direct and consensual and reaction to accommodation.

A Relative Afferent Pupillary Defect was assessed using the swinging flashlight test of Levatin, This was performed in all cases with a standard illumination time on the eye of three seconds, before rapidly swinging the light on the follow eye.

RAPD was graded as follows,

Grade I - A weak initial contraction followed by greater redilatation.

II - Immediate pupillary dilatation

III - Immediate pupillary dilatation following prolonged illumination of good eye for 6 seconds.

IV - Immediate pupillary dilatation with no secondary constriction .

Corneal sensation was looked for with the aid of a wisp of cotton.

Visual acuity for distance was recorded with snellen's charts with and without glass correction (if the patient was already wearing glasses) or with pinhole. Near vision was documented using reduced snellen's test type charts.

Color vision was tested with standard Ishihara chart.

Fundus – Detailed examination of the fundus was done after dilating the pupil with a short acting mydriatic. Details of the optic disc (size, shape, color, margins, c:d ratio) vessels and foveal reflex were noted with direct, Indirect ophthalmoscopic and 90 diopter lens.

Intra Ocular pressure was recorded using applanation tonometer, and Schiotz (Indentation) tonometer.

Charting of the fields was done using Bjerrum's Screen (1 m chart) and by finger confrontation method. Few cases which failed to show a field defect in Bjerrum's but which had a great degree of clinical suspicion were subjected to automated Perimetry (Humphrey).

Proptosis, when present was documented with Hertel's exophthalmometer and all the related parameters (bruit / reducibility / compressibility etc.,) were looked for.

Diplopia chart was done using red-green goggles. The type of diplopia was charted out to help in reviewing the patient as to progression / regression of the complaint.

Examination of Other Systems

Any relevant past history or family history related to diabetes or hypertension was recorded. General status of the patient including cardiac and respiratory functions were assessed. Evaluation of possible endocrine disturbances was done.

Investigations.

Neurological Investigations

After screening the patients, the cases slelected for the study were subjected to X-ray skull, CT scan brain, (plain and contrast) and MRI to confirm and correlate the presence of intracranial tumor.

Ophthalmic Investigations

X-rays of orbit and of both optic foramina were taken to delineate involvement of superior orbital fissure and optic canal.

Fundus Fluorescein Angiography was done in selected cases (after injecting 3 ml of 20% fluorescein intravenously) to confirm the diagnosis of Papilledema when in doubt.

Management

Conservative management included Antibiotics, steroids, and NSAIDs to reduce the inflammatory edema and pain.

Surgical intervention was at the discretion of the neurosurgeon. It was either a total excision or a subtotal excision and biopsy.

Biopsy material was subjected to histopathological examination and further treatment was planned according to the tumor type.

Radiotherapy was given to patients whose tumors were inaccessible surgically or those who had recurrences after a primary excision.

Patients were reviewed periodically and any improvement / deterioration of their visual parameters was noted.

All the above data was recorded on proforma.

ANALYSIS AND DISCUSSION

ANALYSIS & DISCUSSION

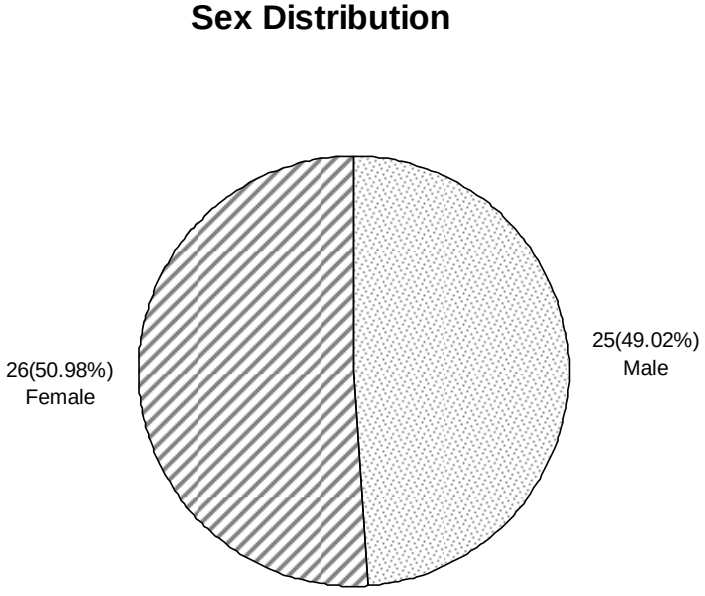
Sex Distribution

Table No : 1

Total	Male	Female
51	25(49.02%)	26 (50.98%)

Out of the 51 patients studied, 25 (49.02%) were male and 26 (50.89%) were female. Hence in our study no sex predilection was noted for brain tumors on the whole.

Primary brain tumors are known to be more common among males[52]. The almost equal occurrence of males and females in our study can be due to the fact that this study was conducted at a tertiary level referral hospital and hence does not actually correlate with a true incidence among the general population.

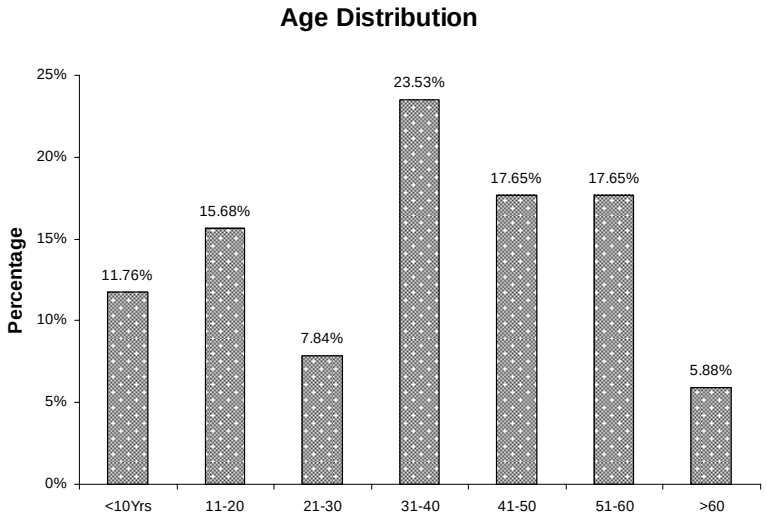


Age Distribution

Table No : 2

Age(years)	No. of patients	Percentage
<10	6	11.76%
11-20	8	15.68%
21-30	4	7.84%
31-40	12	23.53%
41-50	9	17.65%
51-60	9	17.65%
>60	3	5.88%
	51	

In this study population, the maximum number of patients (12) were in the 31-40 year age group. The next most affected age group were in the 41-50 and 51-60 years age group with nine patients in each. Quite a substantial number of patients were in the 41-60 year age group (35.30%). This increased incidence in the elderly population could be due to the better diagnostic techniques available or to a genetic susceptibility to primary malignant brain tumors. There were three patients above the age of 60 in our study



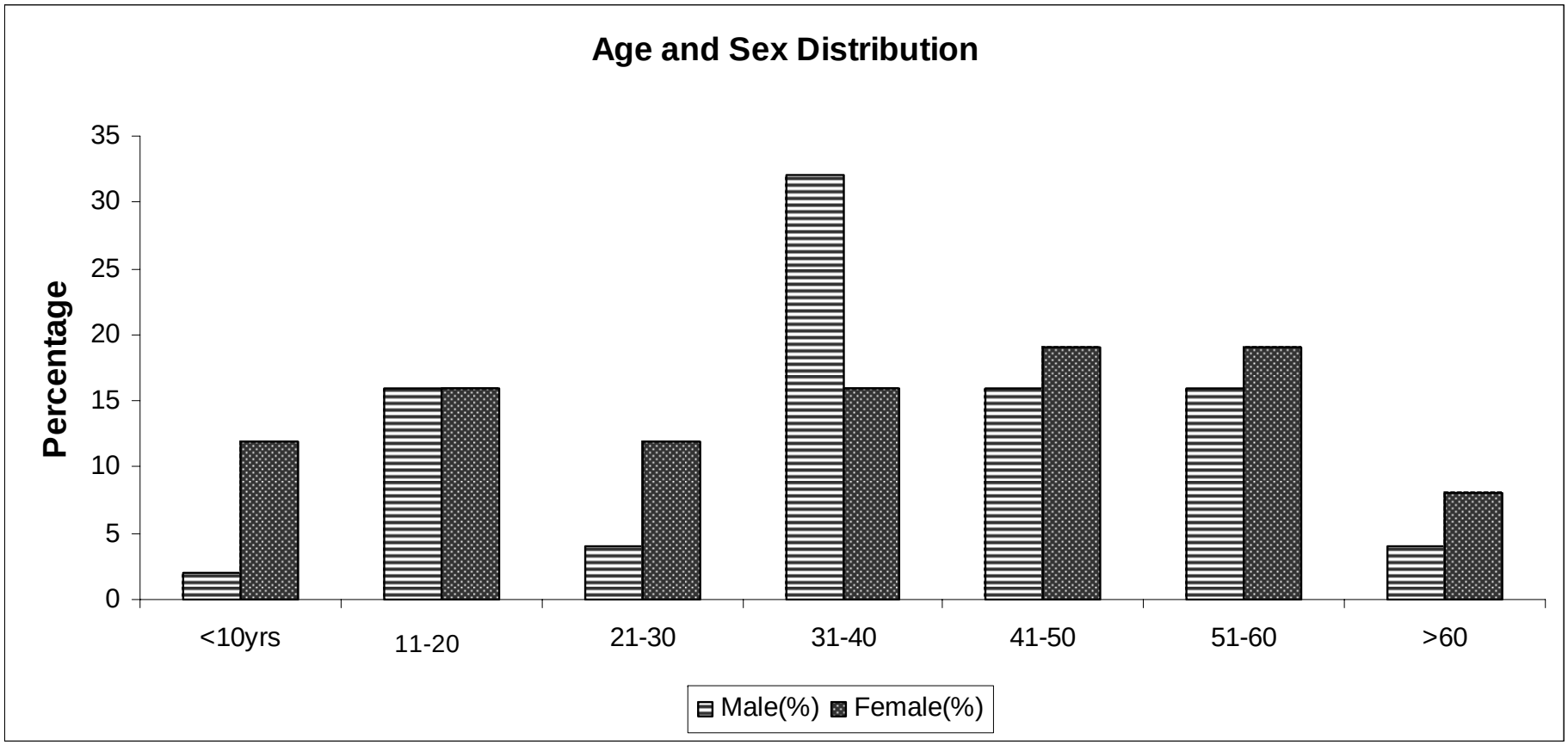
Age and Sex Distributions

Table No : 3

Age(years)	Male(%)	Female(%)	Total
<10yrs	3(2%)	3(11.54%)	6(11.76%)
11-20	4(16%)	4(15.38%)	8(15.68%)
21-30	1(4%)	3(11.54%)	4(7.84%)
31-40	8(32%)	4(15.38%)	12(23.53%)
41-50	4(16%)	5(19.23%)	9(17.65%)
51-60	4(16%)	5(19.23%)	9(17.65%)
>60	1(4%)	2(7.69%)	3(5.88%)
	25	26	51

Males and females were equally affected in both under 10 years and 11-20 years age group (3 and 4 patients respectively). There were more no. of male patients (32%) in the 31-40 year age group than female patients (15.38%). This could be because, our study included people from lower socio economic background where in women do not seek prompt assistance for their problems.

This correlates with the results from Central Brain Tumor Registry of the United States (1990-94), that show a higher incidence of tumors in male patients (12.1 per 1,00,000 person-years) than in female patients (11.0 per 1,00,000 person – years)


Age and Sex Distribution
Percentage
35
30
25
20
15
10
5
0
<10yrs
11-20
21-30
31-40
41-50
51-60
>60
Male(%)
Female(%)

Type of Tumor (Based on HPE Report)

Table No : 4

Type of Tumor	No. of patients	Percentage
Meningioma	6	11.76%
Acoustic neuroma	4	7.84%
Glioma	13	25.49%
Pituitary adenoma	4	7.84%
Craniopharyngioma	6	11.76%
Ependymoma	1	1.96%
Hemangioblastoma	1	1.96%

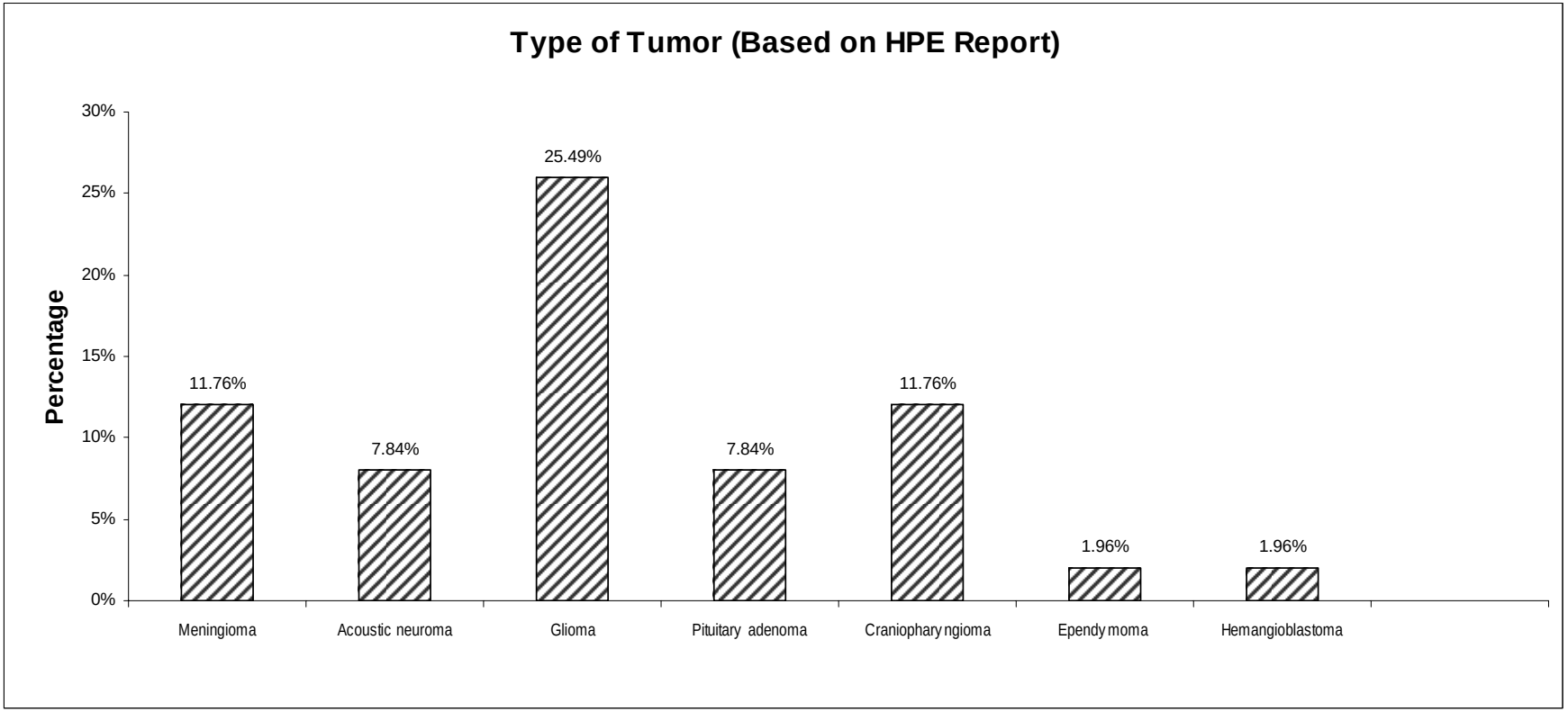
Gliomas formed the major chunk of intracranial tumors in our study with 13 patients (25.49%) belonging to this category.

Meningiomas and Craniopharyngiomas were the next in frequency with 6 patients (11.76%) in each.

Acoustic neuromas and pituitary adenomas were the third in frequency with 4 patients (7.84%) in each.

16 patients (31.37%) were not subjected to excision bipsy of tumor and HPE. Hence these could not be classified among any group.

This correlates well with the data that the glial tumors account for the majority of primary brain tumors (50-60%), followed by meningiomas (25%)[52].

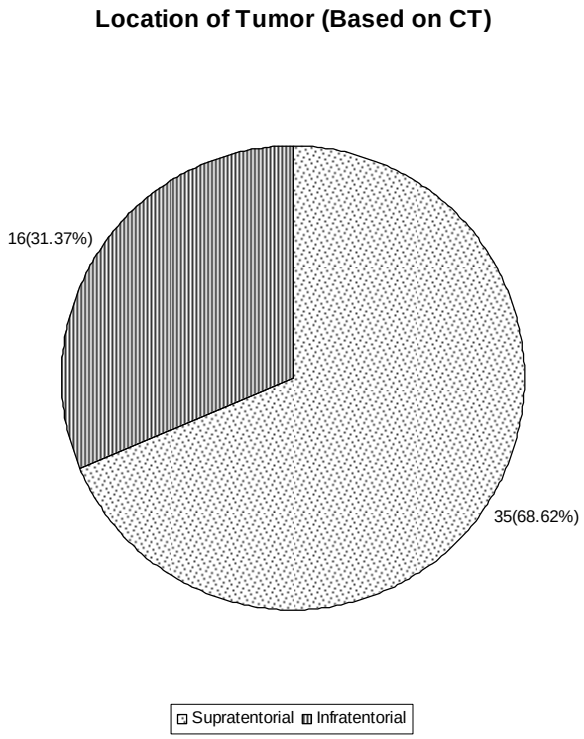

Type of Tumor (Based on HPE Report)
Percentage
30%
25%
20%
15%
10%
5%
0%
11.76%
7.84%
25.49%
7.84%
11.76%
1.96%
1.96%
Meningioma
Acoustic neuroma
Glioma
Pituitary adenoma
Craniopharyngioma
Ependymoma
Hemangioblastoma

Location of Tumor (Based on CT)

Table No : 5

Total	Supratentorial	Infratentorial
51	35(68.62%)	16(31.37%)

Out of the 51 patients, 35 patients (68.62%) had supratentorial tumors and 16 patients (31.37%) had tumors in the infratentorial compartment. In contrast to tumors in the supratentorial region, these infratentorial tumors predominantly manifested with ocular motor disturbance.



Sex Distribution among Different types of Tumors

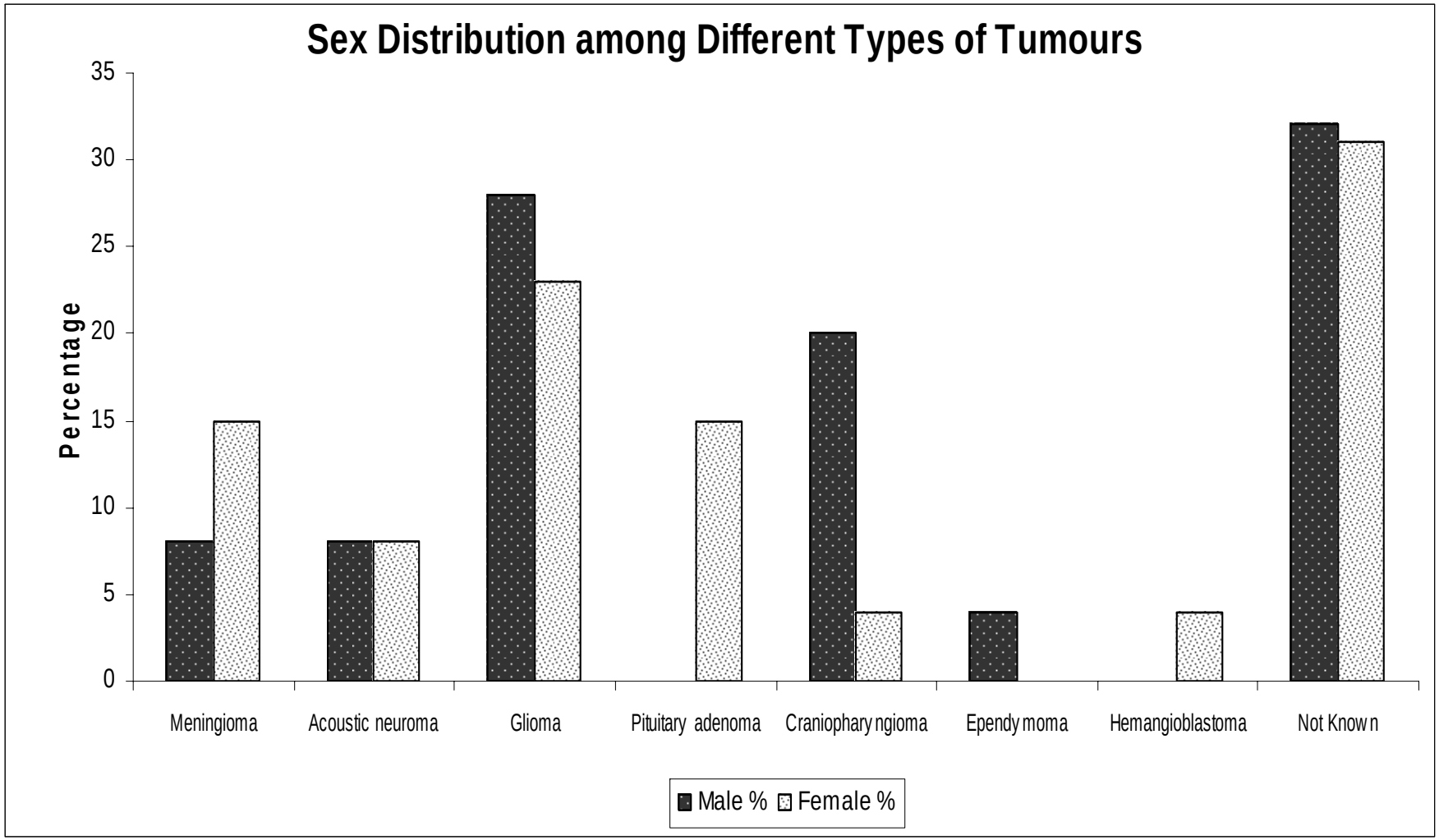
Table No : 6

Type of Tumor	Male	Female	Total
Meningioma	2(8%)	4(15.38%)	6(11.76%)
Acoustic neuroma	2(8%)	2(7.69%)	4(7.84%)
Glioma	7(28%)	6(23.08%)	13(25.49%)
Pituitary adenoma	-	4(15.38%)	4(7.84%)
Craniopharyngioma	5(20%)	1(3.85%)	6(11.76%)
Ependymoma	1(4%)	-	1(1.96%)
Hemangioblastoma	-	1(3.85%)	1(1.96%)
Not Known	8(32%)	8(30.77%)	16(31.37%)

Comparing the incidence of different types of tumors (Based on HPE report) in both the sexes, it was found that meningiomas were seen more frequently in females (4 patients – 15.38%) than males (2 patients – 8%). Acoustic neuromas were found with equal frequency in both males and females.

This correlates well with reports in the literature that meningiomas and Acoustic neuromas are more frequent in women.[51].

Gliomas were found to have a slightly male preponderance (7 patients – 28%) as compared to females. (6 patients – 23.08%)


Sex Distribution among Different Types of Tumours
Percentage
35
30
25
20
15
10
5
0
Meningioma
Acoustic neuroma
Glioma
Pituitary adenoma
Craniopharyngioma
Ependymoma
Hemangioblastoma
Not Known
Male %
Female %

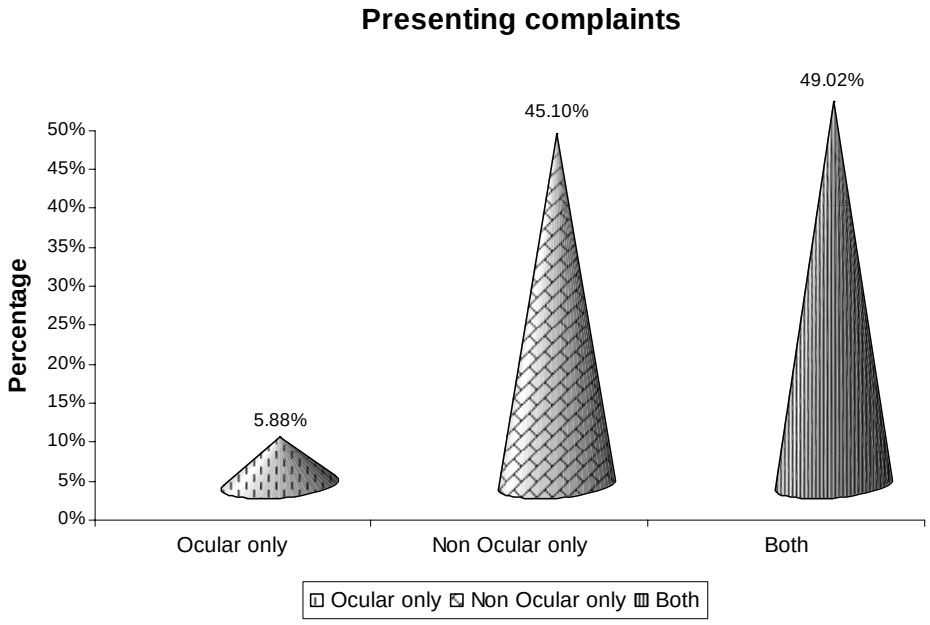
Presenting complaints

Table No : 7

Presenting Complaints	No. of patients	Percentage
Ocular only	3	5.88%
Non Ocular only	23	45.10%
Both	25	49.02%
	51	

Most of the patients presented with both ocular and general symptoms – about 50% of the study group.

There were 3 patients (5.88%) who presented exclusively with ocular symptoms only and hence were bound to be seen by an ophthalmologist first. Two patients presented with complaints of defective vision and one presented with ptosis. Hence ophthalmologists play a major role in recognizing brain tumors at an early stage.



Ocular Symptoms

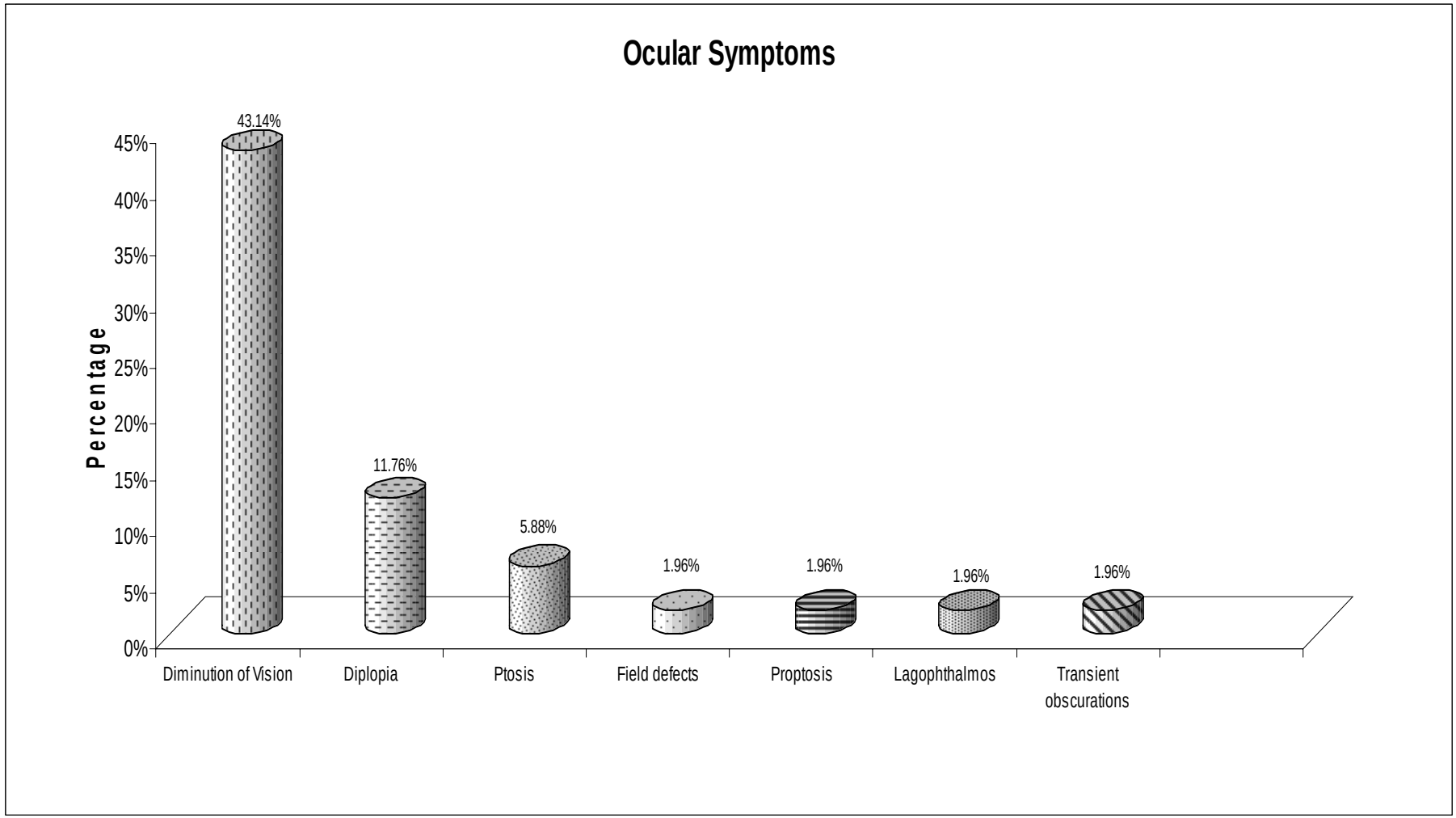
Table No : 8

	No. of patients	Percentage
Diminution of Vision	22	43.14%
Diplopia	6	11.76%
Ptosis	3	5.88%
Field defects	1	1.96%
Proptosis	1	1.96%
Lagophthalmos	1	1.96%
Transient obscurations Of vision	1	1.96%

Out of the many visual problems, diminution of vision was the most common, affecting 22 patients (43.14%). Diplopia was the second most common complaint, mainly due to the ocular motor nerve abnormalities. This correlated with the involvement of sixth cranial nerve which occurred as a “false localizing sign” in patients with increased intracranial pressure.

Typical complaint of Transient obscuration of vision (suggestive of papilledema) was volunteered by 2 patients (3.17%) only, out of the 26 patients (50.98%), who were found to have papilledema on fundus examination. This could be because, our study involved illiterate people from a poor socioeconomic status who might not have noticed the transient obscurations of vision.

Out of the 20 patients, who were found to have defective visual field on examination, only one patient, complained of a field defect. Hence 19 patients were not aware of their field defect, till they were examined.


Ocular Symptoms
Percentage
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
43.14%
11.76%
5.88%
1.96%
1.96%
1.96%
1.96%
Diminution of Vision
Diplopia
Ptosis
Field defects
Proptosis
Lagophthalmos
Transient obscurations

Correlation between Ocular Symptoms and site of brain tumor

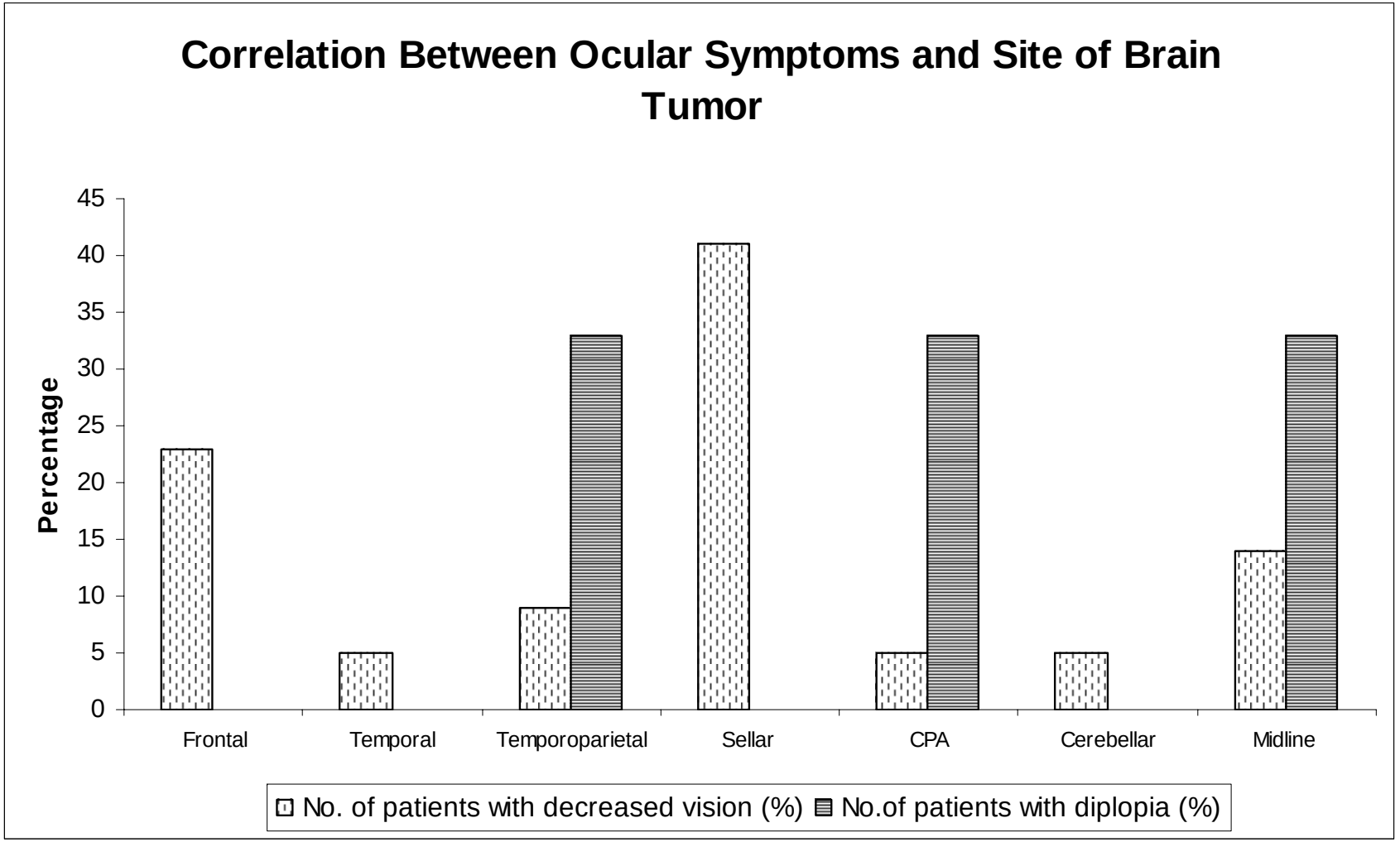
Table No : 9

Site of brain Tumor	No. of patients with decreased vision (%)	No.of patients with diplopia
Frontal	5(22.73%)	-
Temporal	1(4.55%)	-
Temporoparietal	2(9.09%)	2(33.33%)
Sellar / Parasellar Suprasellar	9(40.91%)	-
CPA	1(4.55%)	2(33.33%)
Cerebellar	1(4.55%)	-
Midline (Parasagittal Posterior fossa)	3(13.64%)	2(33.33%)
	22	6

Out of the 22 patients, who complained of diminished visual acuity, 9 patients (40.91%) had a space occupying mass in the sellar, suprasellar or parasellar region. These space occupying lesions were later confirmed to be pituitary adenomas and Craniopharyngiomas by HPE. This finding is in concordance with the anatomy showing that any sellar mass is bound to cause a compressive damage to the optic chiasm and hence causes a lowering of visual acuity.

As a general rule, the two earliest and outstanding symptoms are headache and failing vision so that many patients see the ophthalmologist first; his responsibility for early diagnosis is therefore great[48].

Out of the 6 patients who complained of diplopia, majority also had papilledema. Hence diplopia can be accounted for by the sixth cranial nerve palsy due to increased intracranial pressure (false localizing sign).


Correlation Between Ocular Symptoms and Site of Brain Tumor
Percentage
45
40
35
30
25
20
15
10
5
0
Frontal
Temporal
Temporoparietal
Sellar
CPA
Cerebellar
Midline
No. of patients with decreased vision (%)
No.of patients with diplopia (%)

Non Ocular Symptoms

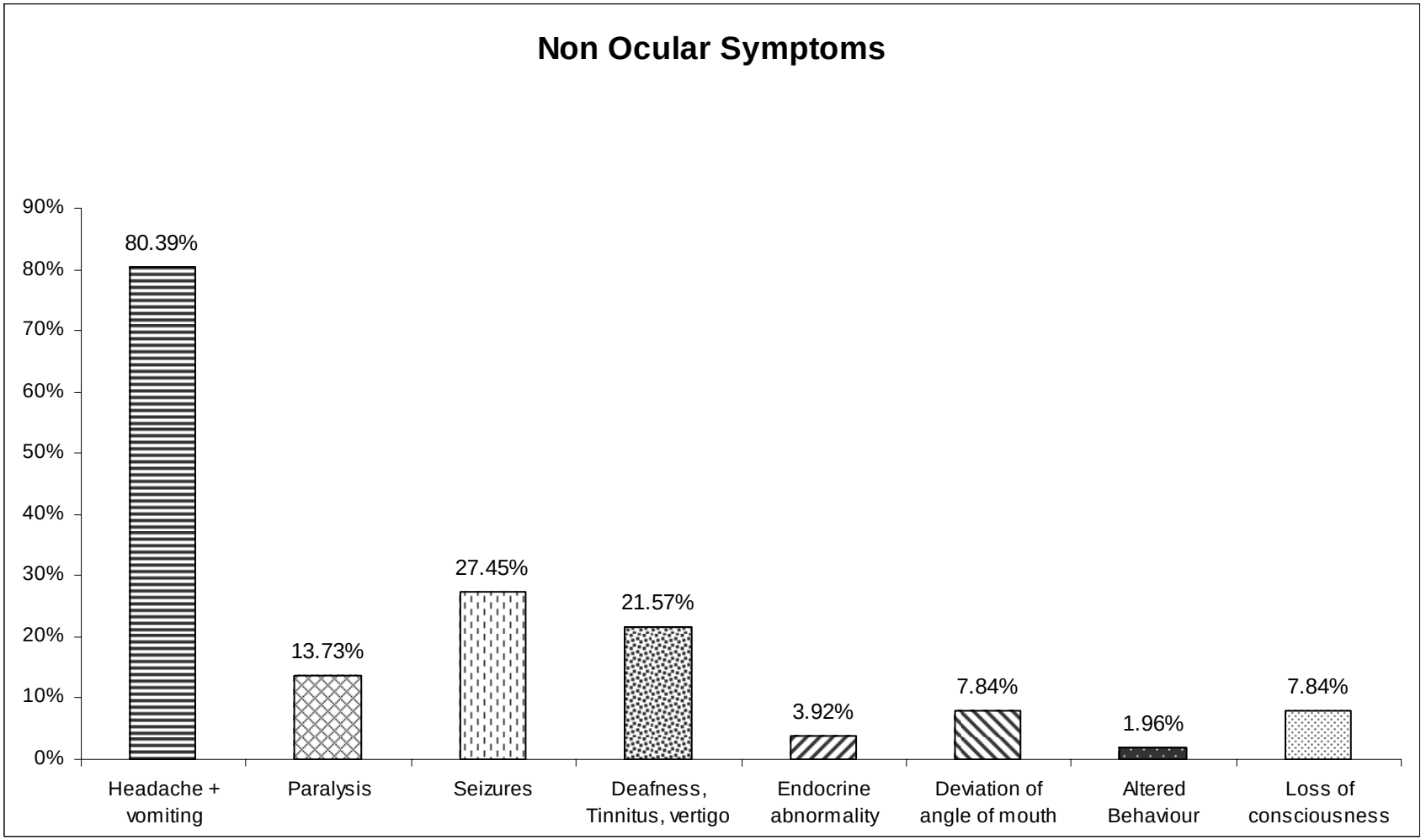
Table No : 10

Non ocular symptoms	No. of Patients	Percentage
Headache + vomiting	41	80.39%
Paralysis	7	13.73%
Seizures	14	27.45%
Deafness, Tinnitus, vertigo	11	21.57%
Endocrine abnormality	2	3.92%
Deviation of angle of mouth	4	7.84%
Altered Behaviour	1	1.96%
Loss of consciousness	4	7.84%

Headache (with or without vomiting) was the single most consistent symptom occurring in 41 patients (80.39%). This correlates well with the studies according to Rushton and Rooke, 1962 and the childhood brain tumor consortium, 1991, where headache was seen to occur in 75% or more of patients. Even when other complaints were present, headache was often told to be the first symptom by the patients in this study.

Headache was seen to occur in almost all the patients irrespective of the site of tumor. Neither severity of headache nor its location has any real value in localization of cerebral tumor[50].

Motor abnormalities including weakness / paresis of limbs or gait disorders were also frequently complained of (13.73%). Seizures whether focal or diffuse type accounted for another significant reason for seeking medical attention (27.45%)


Non Ocular Symptoms
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
80.39%
13.73%
27.45%
21.57%
3.92%
7.84%
1.96%
7.84%
Headache + vomiting
Paralysis
Seizures
Deafness, Tinnitus, vertigo
Endocrine abnormality
Deviation of angle of mouth
Altered Behaviour
Loss of consciousness

Correlation between Neurological symptoms and site of Tumor

Table No : 11

Site of brain tumor	Weakness / Paralysis	Seizures	Tinnitus, Vertigo Deafness
Frontal	-	4(28.57%)	1(9.09%)
Temporal	1(14.29%)	2(14.29%)	1(9.09%)
Frontoparietal	1(14.29%)	1(7.14%)	-
Temporoparietal	1(14.29%)	3(21.43%)	-
Parietal	1(14.29%)	1(7.14%)	1(9.09%)
Sellar / parasellar/ Suprasellar	1(14.29%)	1(7.14%)	-
CPA	-	-	7(63.64%)
Cerebellar	-	1(7.14%)	1(9.09%)
Midline (Parasagittal, Posterior Fossa)	2(28.57%)	1(7.14%)	-
	7	14	11

Among the 7 patients, who complained of weakness or paralysis, 4 patients (57.16%), had a mass in the vicinity of parietal and temporal lobes. This is in accordance with the involvement of pyramidal tract in these areas, and is mostly associated with a UMN type of VII Cranial nerve palsy.

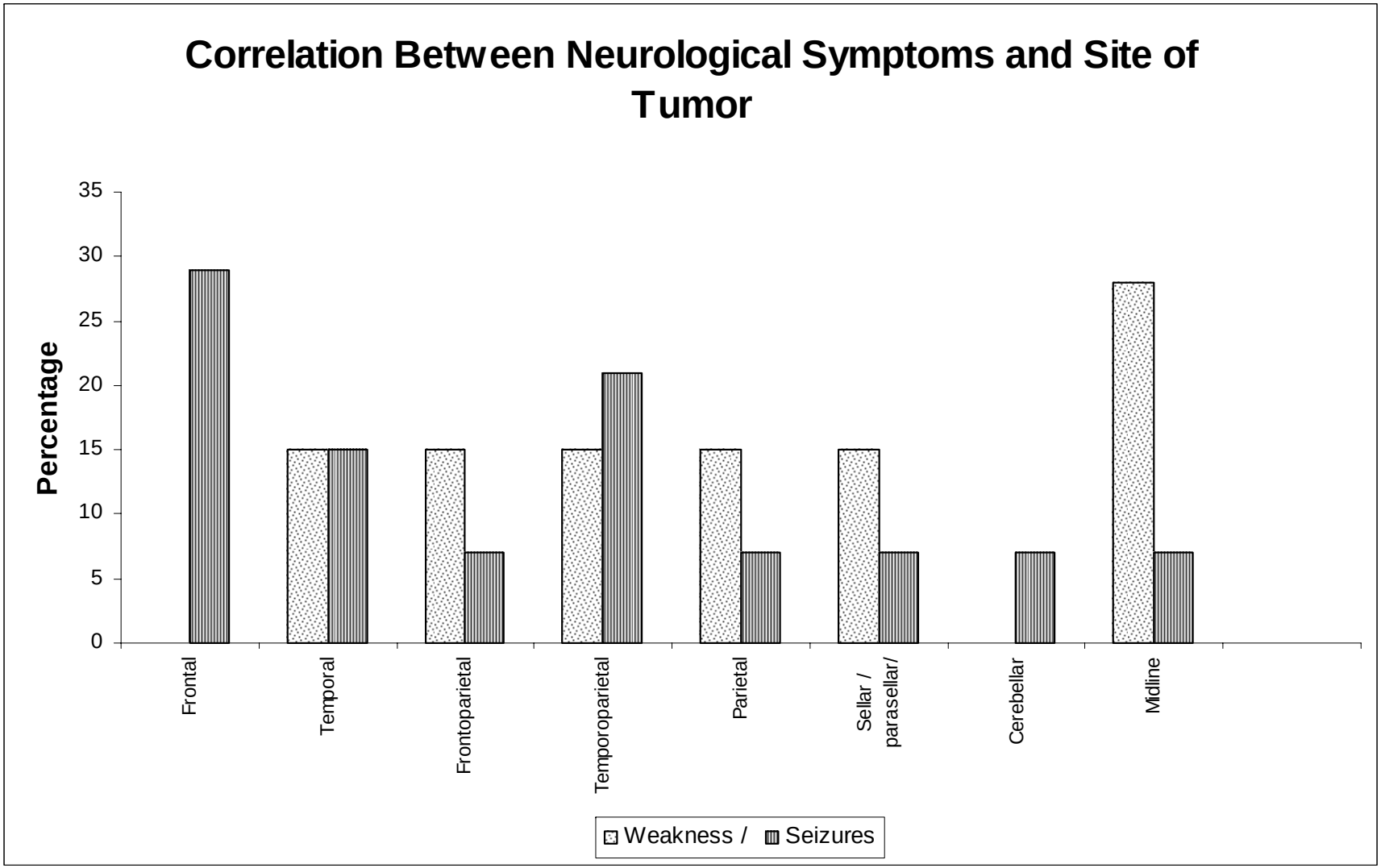
Convulsions were seen to occur in 5 patients (35.71%) with lesions in and around parietal lobe (Parietal and Temporoparietal Mass). The occurrence of

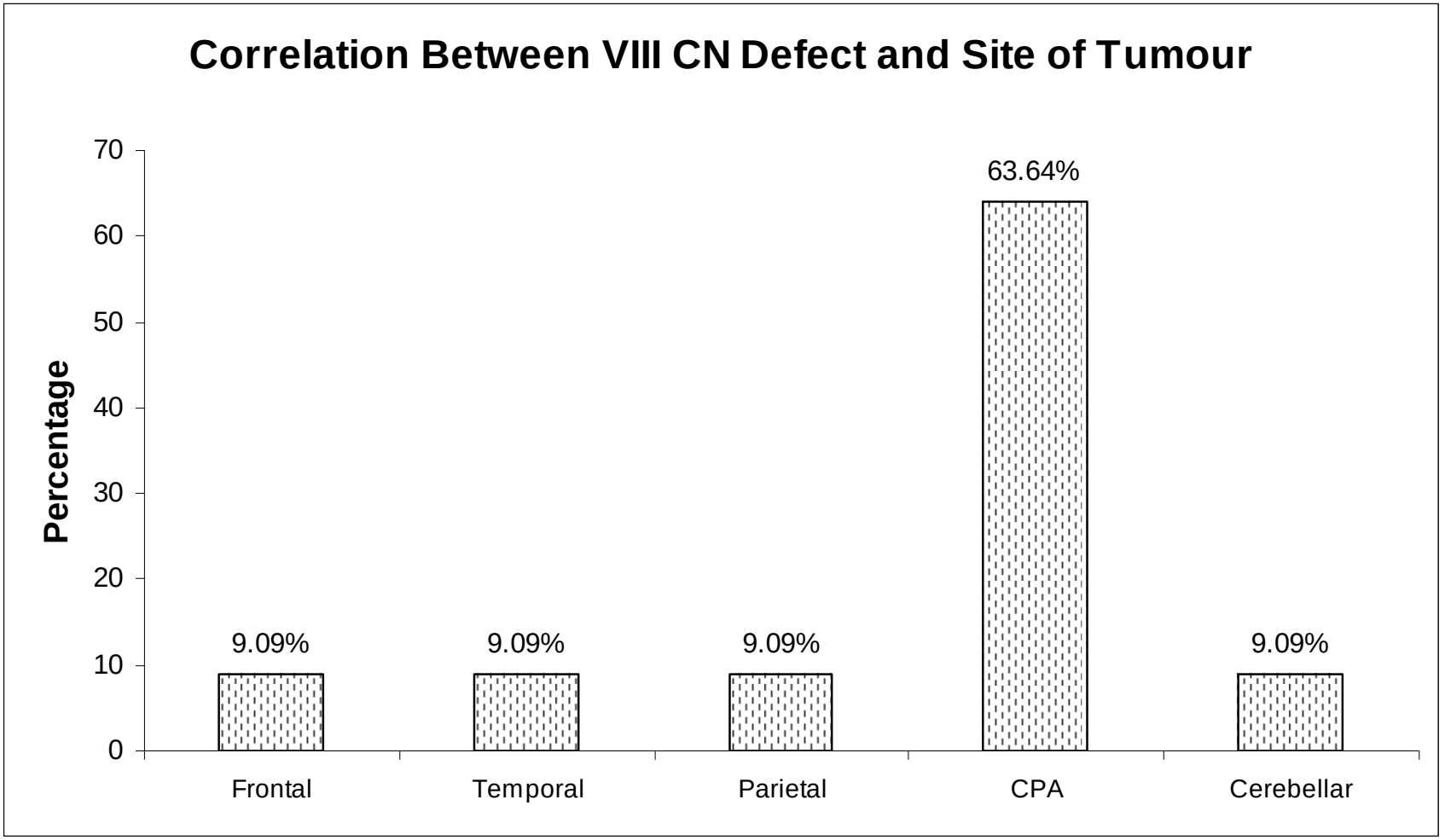
seizures due to a mass in this region is usually accompanied by visual hallucinations and aura (Forsyth & Posner, 1993)

28.57% of patients with seizures, were found to have tumors in the region of the frontal lobe.

Head turning and eye turning are accompanied by slow tonic changes in the position of the arm and sometimes the leg in the direction towards which the eyes deviate. Following the seizure, the eyes may be directed toward the side of the tumor as part of a postictal paralysis of function [45].

Involvement of VIII Cranial nerve causing deafness or tinnitus or vertigo occurred in 11 patients out of which 7 patients (63.64%) had a CPA mass. Hence any complaint of defective hearing in a patient suspected to harbour a brain tumor should be immediately investigated for a CPA tumor.


Correlation Between Neurological Symptoms and Site of Tumor
Percentage
35
30
25
20
15
10
5
0
Frontal
Temporal
Frontoparietal
Temporoparietal
Parietal
Sellar / parasellar/
Cerebellar
Midline
Weakness /
Seizures


Correlation Between VIII CN Defect and Site of Tumour
Percentage
70
60
50
40
30
20
10
0
9.09%
9.09%
9.09%
63.64%
9.09%
Frontal
Temporal
Parietal
CPA
Cerebellar

Ophthalmic Signs

Table No : 12

Ophthalmic signs	No. of patients	Percentage
Papilledema	26	50.98%
II CN	16	31.37%
III CN	1	1.96%
V CN	7	13.72%
VI CN	7	13.72%
III & VI CN	3	5.88%
VII CN	7	13.72%
Field defect	20	39.21%
Nystagmus	5	9.80%
Gaze palsy	1	1.96%
Proptosis	1	1.96%

On examining the patients, papilledema was found to be the major clinical sign occurring in 26 patients (50.98%). Though this formed the most common ophthalmic sign, the occurrence is less than studies done elsewhere. Papilledema occurred in 80% of cases (Gowers, 1904; Paton, 1909;) and 77.9% of a total 1239 cases (Tonnis and krenkel, 1957)[50].

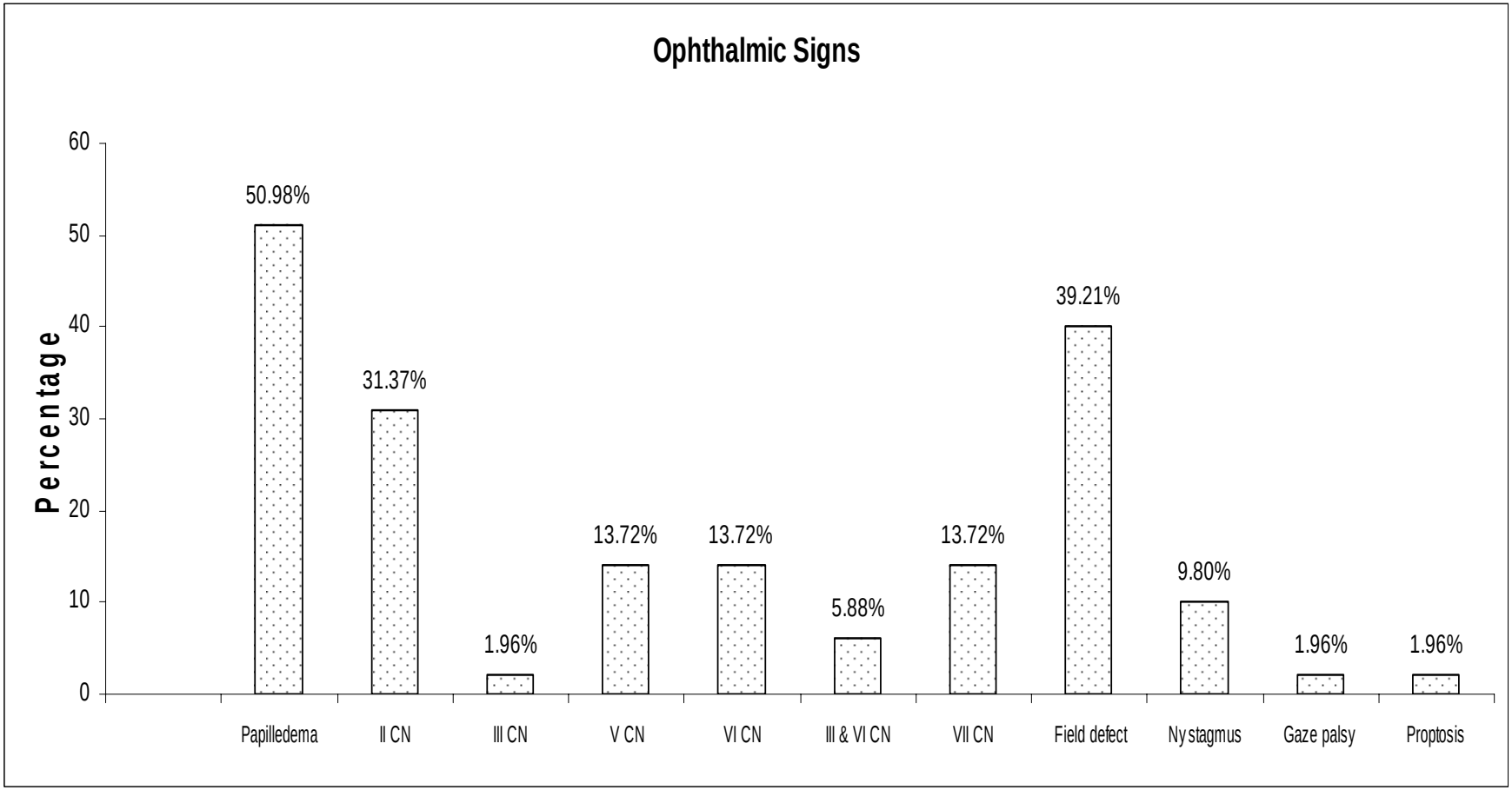
In our study FFA was done only in few cases with a clinical suspicion of papilledema. Probably routine FFA for all patients would yield a higher incidence of papilledema. Papilledema may be observed at almost any age. There is no upper limit and may be present even in infants and children[50].

The next frequent manifestation was of cranial nerve palsies out of which optic nerve was most commonly affected. (16 Patients – 36.37%)

Optic atrophy of some degree is a common result of pituitary tumors, although it is by no means invariable, particularly when the growth is postchiasmal (Mc Connell and Mooney, 1938). The pallor is largely due to ischemia and this appearance may not necessarily be associated with a loss of function until the nerve fibres themselves are affected [48].

Out of the cranial nerves governing ocular motility, sixth cranial nerve palsy isolated (or) along with other ocular motor nerves, was observed in majority (10 patients – 19.6%). Field defects were seen in 20 patients (39.21%) which had a great localizing value and aided the topical diagnosis of tumors profoundly.

Nystagmus was observed in 5 patients (9.80%) of whom, 4 were found to have a CP angle tumor.


Ophthalmic Signs
Percentage
60
50
40
30
20
10
0
50.98%
31.37%
1.96%
13.72%
13.72%
5.88%
13.72%
39.21%
9.80%
1.96%
1.96%
Papilledema
II CN
III CN
V CN
VI CN
III & VI CN
VII CN
Field defect
Nystagmus
Gaze palsy
Proptosis

Correlation of ophthalmic signs with the site of tumor

Table No : 13

	Papilledema	II CN Defect	Eye Movt Defect	Field Defect
Frontal	2(7.69%)	3(18.75%)	1(10%)	-
Frontoparietal	1(3.84%)	-	-	-
Temporal	2(7.69%)	-	2(20%)	2(10%)
Parietal	4(15.38%)	-	1(10%)	-
Temporoparietal	4(15.38%)	-	1(10%)	4(20%)
Sellar/suprasellar Parasellar	-	10(62.5%)	2(20%)	11(50%)
CPA	7(26.92%)	-	2(20%)	2(10%)
Cerebellar	2(7.69%)	-	-	1(5%)
Midline (Parasagittal, Posterior fossa)	4(15.38%)	3 (18.75%)	1(10%)	-
	26	16	10	20

On studying the occurrence of papilledema with tumors of different location, it was found that 7 patients (26.92%) had a CPA mass. Infratentorial tumors that arise from cerebellum and fourth ventricle tend to increase intracranial pressure early by obstructing the flow of CSF through the aqueduct of sylvius [45].

Hartmann et al studied the ocular fundi of 1169 patients with verified intracranial tumor and found that gliomas within brain produced papilledema in 76% of cases. Meningiomas compressed brain in 40% and there were tumors of posterior fossa in 71% of cases[50].

According to Stopford, supratentorial tumors rarely result in primary obstruction to flow of CSF through aqueduct of sylvius and in such tumors, Papilledema is due to deflection of the falx and pressure upon great vein of galen[50].

VP Shunt was performed in 9 patients. The decision was based upon the patients's neurological status, as well as the appearance of the disc, visual acuity and fields.

Indications for intervention in papilledema include Disc swelling of more than 5D, great engorgement of veins, presence of extensive haemorrhages, early appearanceof cytoid bodies, decrease in visual acuity, arterial narrowing and peripheral constriction of visual field [48].

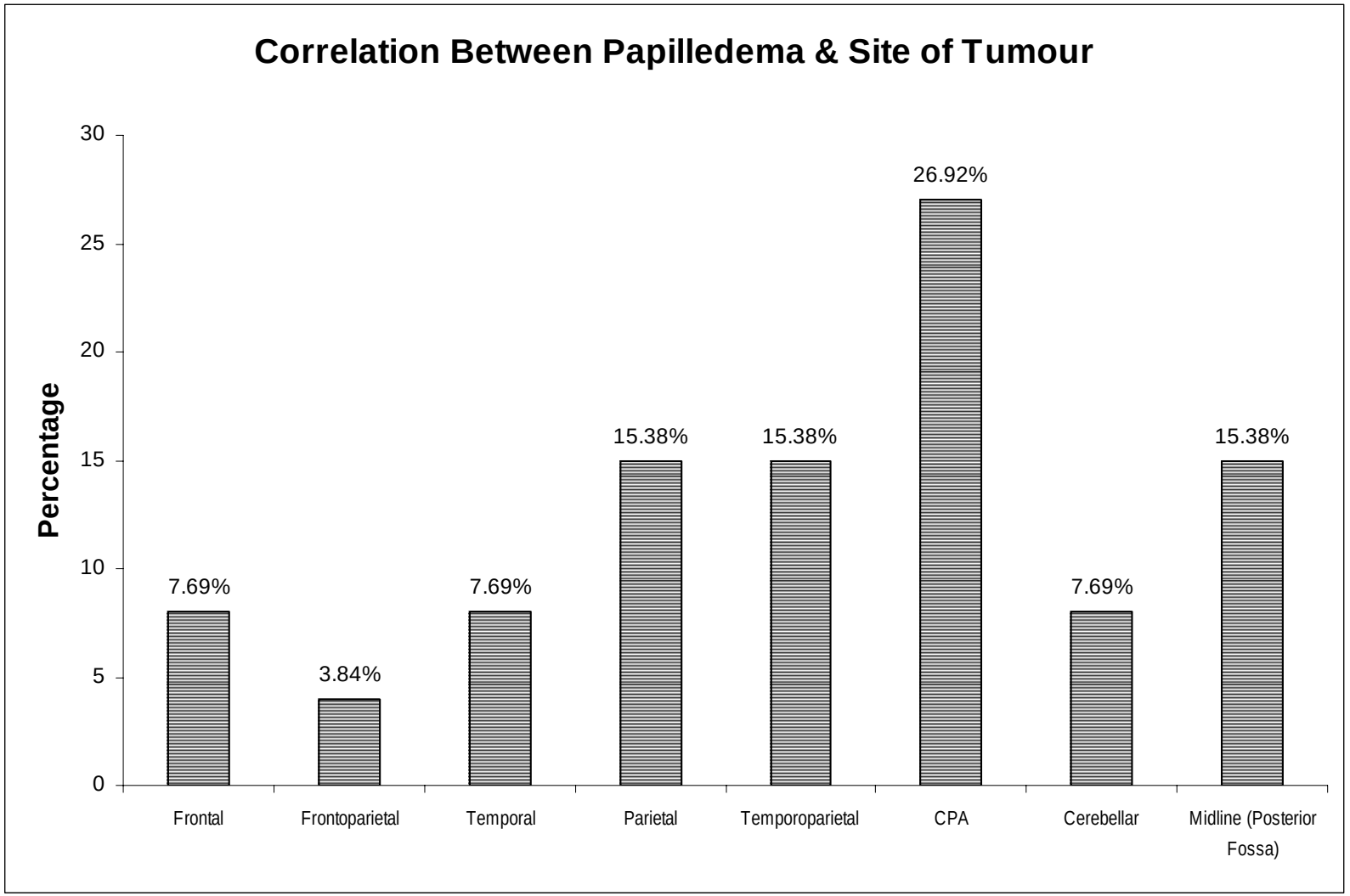
On examining the second cranial nerve with pupillary reaction, visual acuity, color vision and ophthalmoscopy, it was found to be affected in 16 patients (36.37%). Of these 16 patients, 10(62.5%) had a sellar / parasellar mass. Hence any complaint of visual loss should lead the investigation towards finding a lesion in this region. More over in any documented case of sellar mass, visual acuity should be assessed regularly to plan the surgical intervention and predict the prognosis

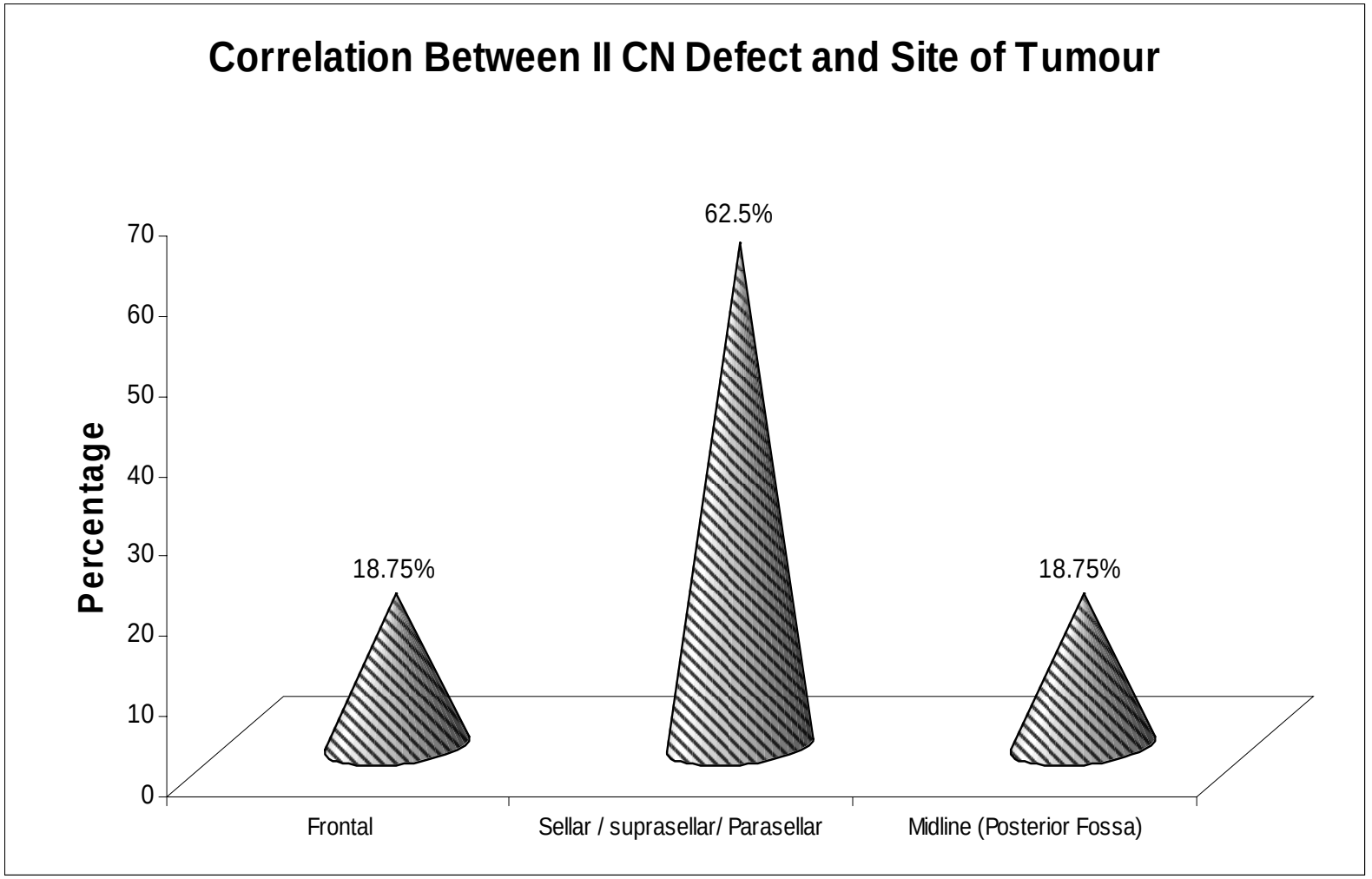
On examining patients with defective visual fields 55% were found to have a sellar / suprasellar mass. Once again this emphasizes the charting of visual fields in every patient suspected to have a brain tumor so as to help in localizing the site of tumor.

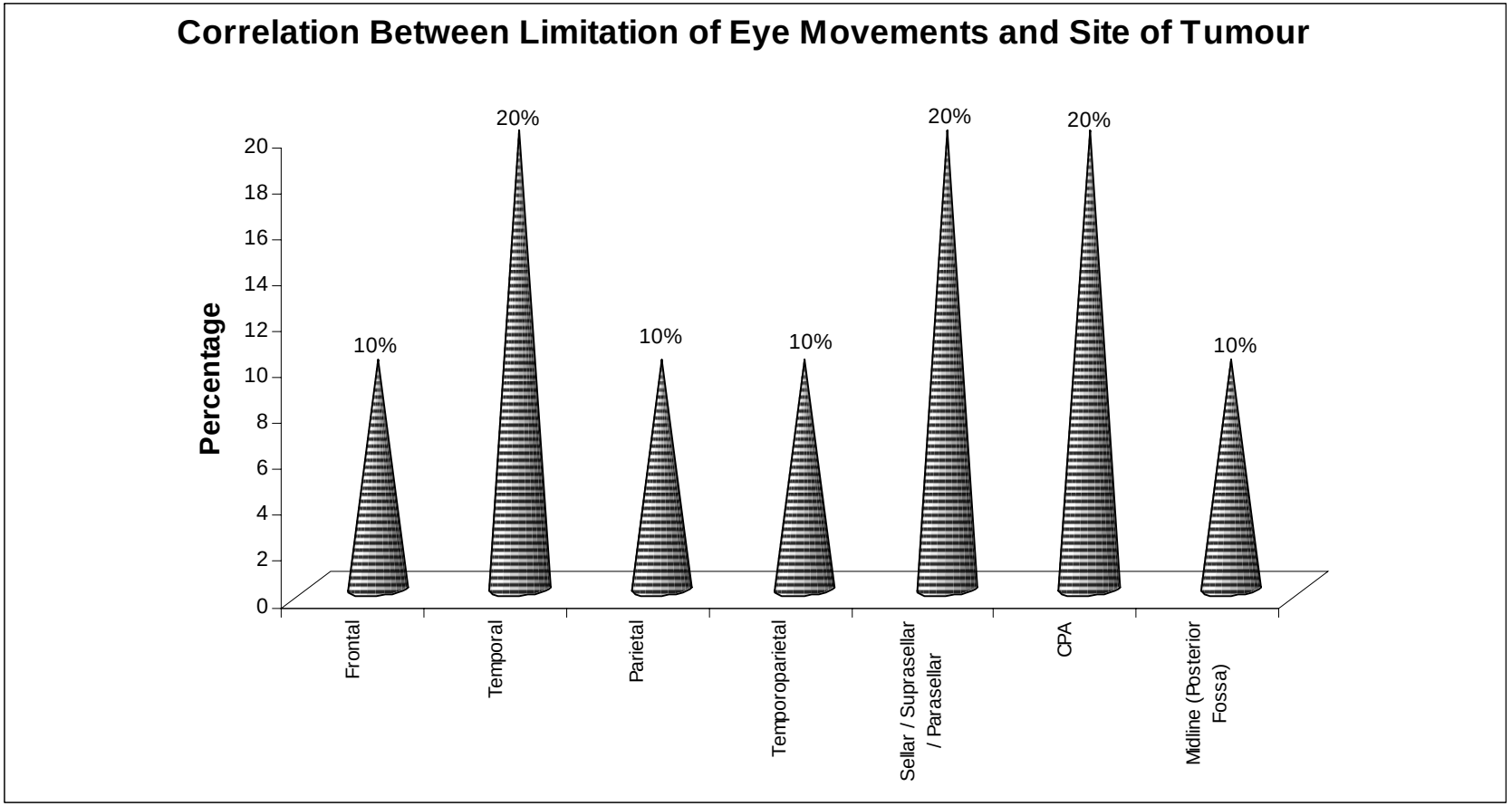
During follow up, the resolution of papilledema was observed in 3 patients, after removal of the intracranial tumor. There was no consistent relation between severity of papilledema and time of resolution.

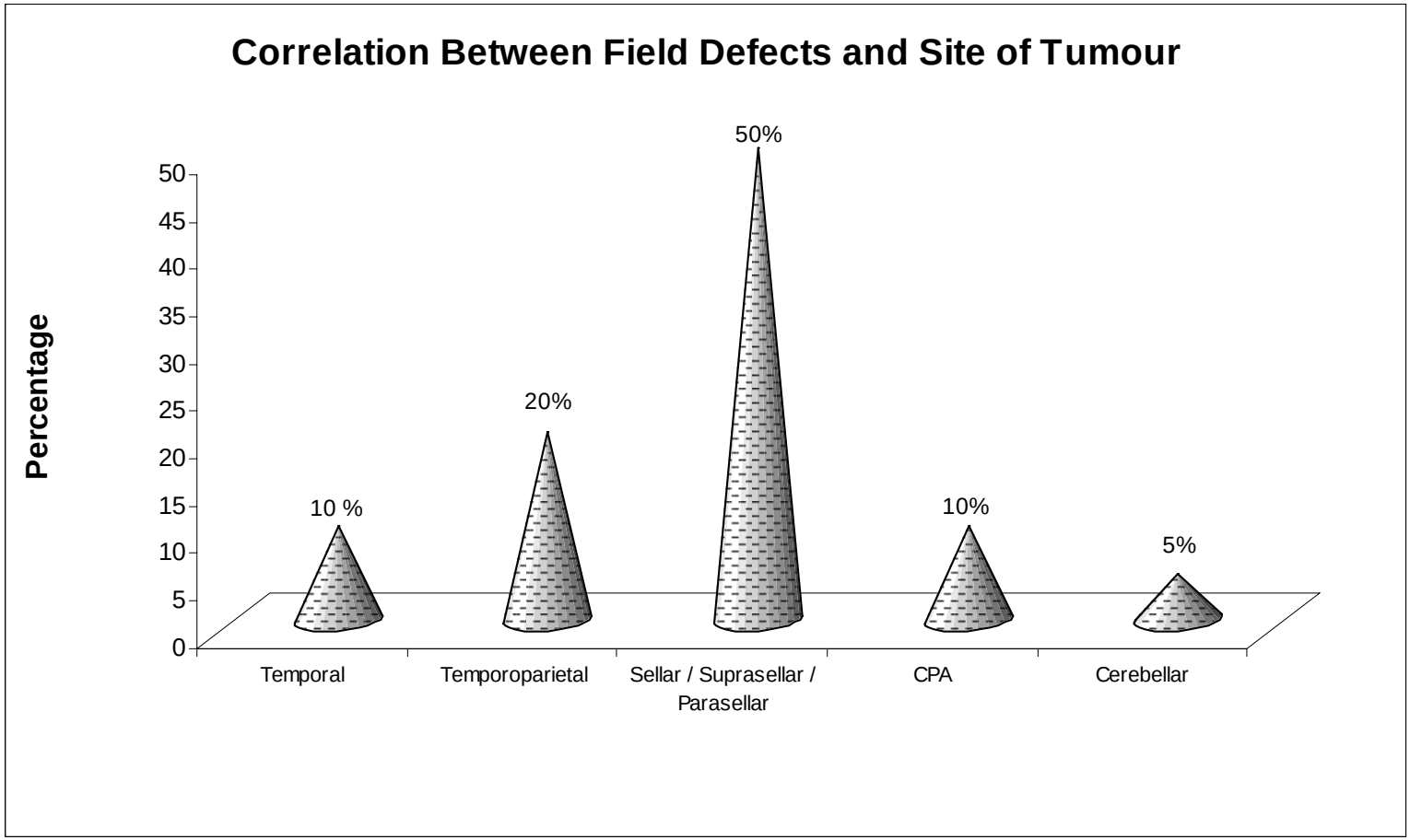
Peristence of papilledema after removal of an intracranial tumor may be explained by raised CSF pressure caused by a postoperative CSF ciculation block which occurs in some patients. If vision is threatened in such cases, a CSF shunt operation should be performed.

Out of the 10 patients who underwent surgical removal of tumors in the area of the optic chiasm, improvement of visual acuity was noted in 3 patients (30%). Visual acuity remained unchanged in 6 patients (60%) and worsening of visual acuity was noted in one patient (10%). Results however depended upon the primary functional and morphological situation. Nevertheless, an appropriate procedure was found to provide excellent prognosis for improvement of visual acuity and fields.


Correlation Between Papilledema & Site of Tumour
Percentage
30
25
20
15
10
5
0
7.69%
3.84%
7.69%
15.38%
15.38%
26.92%
7.69%
15.38%
Frontal
Frontoparietal
Temporal
Parietal
Temporoparietal
CPA
Cerebellar
Midline (Posterior Fossa)


Correlation Between II CN Defect and Site of Tumour
Percentage
70
60
50
40
30
20
10
0
62.5%
18.75%
18.75%
Frontal
Sellar / suprasellar/ Parasellar
Midline (Posterior Fossa)


Correlation Between Limitation of Eye Movements and Site of Tumour
Percentage
0
2
4
6
8
10
12
14
16
18
20
10%
20%
10%
10%
20%
20%
10%
Frontal
Temporal
Parietal
Temporoparietal
Sellar / Suprasellar / Parasellar
CPA
Midline (Posterior Fossa)


Correlation Between Field Defects and Site of Tumour
Percentage
50
45
40
35
30
25
20
15
10
5
0
10 %
20%
50%
10%
5%
Temporal
Temporoparietal
Sellar / Suprasellar / Parasellar
CPA
Cerebellar

SUMMARY

SUMMARY

- The clinical study was conducted at the Coimbatore medical college hospital, Coimbatore during the period February 2004 to March 2006.

- Out of the 65 patients screened, 51 were finally selected based on the inclusion criteria.

- There were 25 male patients (49.02%) and 26 female patients (50.98%).

- The maximum incidence of brain tumors was seen in the 31-40 years age group (12 patients – 23.53%). Sex distribution was equal in patients under age of 20 years. In the 31-40 years age group there were more number of male patients (32%) than female patients (15.38%).

- CT Scan was the main diagnostic tool used to confirm the presence or absence of an intracranial mass.

- Gliomas were the single most common type of tumor (13 patients - 25.49%) based on the HPE report.

- Meningiomas and craniopharyngiomas were the next in frequency.

- Headache was the major presenting complaint occurring in 41 patients (80.39%).

- Gradual loss of vision was the most prominent ocular complaint (43.14%) suggesting a compressive effect on the visual pathway.

- Papilledema was the salient ocular manifestation and it was always associated with increased intracranial pressure caused due to the mass effect of brain tumors. Fundus Fluorescein Angiography assisted in diagnosis of papilledema.

- Ocular muscle palsies were dominated with sixth cranial nerve involvement, which served as a warning sign for brain tumors and occurred as a false localizing sign.

- Involvement of II cranial nerve causing either a fall in vision or a field defect was found to be common in sellar lesions causing compression of optic chiasm.

- Out of 11 patients with VIII nerve involvement 7 patients (63.64%) were found to harbour a CP Angle mass.

CONCLUSION

CONCLUSION

- Among the 51 patients studied 31-40 year age group was the most commonly affected.

- There was no sex predilection on the whole in this study.

- Glioma was found to be the most common Brain Tumor.

- Headache occurred as a non localizing symptom, being present in almost all the patients irrespective of the location of tumors.

- Loss of vision or a field defect was seen in lesions causing chiasmal compression (sellar, suprasellar or parasellar mass).

- Papilledema was the most striking ophthalmic sign and it was seen to occur in CPA tumors most frequently.

- Sixth cranial nerve palsy occurred as a false localizing sign as a result of raised intracranial pressure.

- About 6% of patients in our study, had presented with ocular symptoms only. Hence ophthalmic manifestations are a predominant feature of intracranial tumors.

- Ophthalmic manifestations provide major clues to diagnosis of the intracranial tumors, as well as follow up of these patients. Irreversible loss of vision is possible if these clues are overlooked which can be

prevented by appropriate treatment. Hence consideration should be given to periodic ophthalmological examination of these patients.

- Ophthalmologists play a pivotal role not only in helping neurosurgeons in decisions regarding surgical intervention and post operative management, but also in diagnosis of intracranial tumors in the earliest stage.

- Ocular signs aided in localizing the site of tumors, especially in association with neurological signs

- In this era of advancing radiology, and costly neuro imaging techniques, ocular signs prove to be the best clinical tool in the diagnosis of intracranial tumors.

- Our study emphasizes the importance of team work and the need for multidisciplinary approach to the early diagnosis, appropriate management and follow up of patients with Intra cranial tumors.

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APPENDIX

PROFORMA

CASE PROFORMA

OPHTHALMIC MANIFESTATIONS OF INTRA CRANIAL TUMOURS

NAME	**:**	**S. NO**	**:**
AGE	**:**	**IP NO**	**:**
SEX	**:**	**UNIT**	**:**
ADDRESS	**:**	**WARD:**	

PRESENTING COMPLAINTS :

HISTORY OF PRESENTING COMPLAINTS

General Complaints :

Headache

Vomiting

Motor Symptoms	-	Weakness Spasticity Seizures – Focal / generalized Aura / Hallucinations
Sensory Disturbances	-	Tingling / Numbness Paresthesias
Mental Changes	-	Withdrawn behaviour / Apathy / Irritability / Depression / Memory Loss. Lack of Judgement / Disorientation / Poor abstract thinking.
Gait Disorder		
Nasal Symptoms	-	Obstruction / Discharge / Bleeding
Failure to thrive.		
Endocrine Abnormalities	-	Enlargement of hands & feet
	-	Prominence of jaw
	-	Alteration of speech (Thickening of tongue)
	-	Decreased libido / Impotence
	-	Amenorrhoea
	-	Galactorrhoea

- Weight gain (Moon face) / Weight loss
- Delayed body growth
- Intolerance to cold / heat
- Polydypsia / Polyuria
- Increased pulse rate
- Increased sweating
- Hot / Moist palms
- Increased skin pigmentation

Cranial nerve abnormalities
- Anosmia
- Dimness of vision
- Diplopia
- Abnormal EOM
- Loss of sensation of one half of face
- Difficulty in Chewing
- Deviation of angle of mouth
- Drooling of saliva on one side
- Accumulation of food on one side
- Inability to close eye properly
- Hearing loss
- Tinnitus
- Vertigo
- Nasal twang of voice
- Nasal regurgitation of food
- Deviation of tongue

Ocular Complaints		**RE**	**LE**
Visual Disturbances	:	Dimness of vision Blunting of colour perception Transient loss of Vision Field Loss, Diplopia	
Protrusion of eyeball	:		
Drooping of upper eyelid	:		
Difficulty in closure of eyelids			
Eye movement abnormality	:		
PAST HISTORY	**:**	DM / HT / Pulm TB / BA / IHD / CVA Any significant illness & Rx taken Any operations undergone	
FAMILY HISTORY	**:**		
MENSTRUAL HISTORY	**:**		

PERSONAL HISTORY :

GENERAL PHYSICAL EXAMINATION :

Consciousness Pulse BP

Built / Nourishment

Pallor / Icterus / Cyanosis / Clubbing / Oedema / Lymphadenopathy

CNS EXAMINATION :

HMF : Consciousness (EMV)
Orientation : Time / Place / Person
Memory : Recent / Short term / Remote
Co-operation :
Behaviour :
Judgement :
Speech : Normal / Slurred / Dysarthria / Dysphasia
Handedness (Cerebral Dominance)
Mood

Cranial Nerves

1. Olfactory
2. Optic
3. Oculomotor
4. Trochlear
5. Trigeminal - Motor
   Sensory – Touch / Pain / Corneal Reflex
6. Abducens
7. Facial - Motor
   Sensory – Taste – ant 2/3 of tongue
8. Acoustic - Weber's test
9. Glossopharyngeal
10. Vagus - Gag reflex / Palatal movements
    Voice / Swallowing
11. Accessory- Shrugging of shoulders
12. Hypoglossal- Deviation of tongue
    Wasting / Fibrillation of tongue

			UL	LL
Motor	**:**	Tone		
		Power		
		DTR		
		Plantar reflex		
		Co-ordination (cerebellar)		
		Involuntary movement		
Sensory		Touch		
		Temperature		
		Pain		
		Vibration		

Skull & Spine :

Cerebellar Signs :

Meningeal Signs :

OCULAR EXAMINATION			**RE**	**LE**
Head Position				
Eye brows				
Eye lids		Ptosis		
		Lid retraction		
		Lagophthalmos		
EOM	-	Ductions		
		Versions		
		Vergences		
		Saccades		
		Smooth Pursuit		
Conjunctiva				
Cornea				
	-	sensation		

AC

Iris

Pupil - Size
- Shape
- Light reflex – DR / CR
- Near reflex

Lens

Fundus

Vision - Distant

- Near

Colour vision

Tension

Fields - Central - Bjerrum's

- Automated Perimetry

Peripheral (Lister's)

Diplopia Charting

Nystagmus

CVS EXAMINAION : Heart rate
Heart Sounds
Rhythm
Carotids – Pulsation / Bruit

RS EXAMINATION **:** RR
Nature
Trachea
Breath Sounds

ABDOMINAL EXAMINATION : Hepatomegaly / Splenomegaly / Mass
Bowel Sounds

SUMMARY :

PROVISIONAL DIAGNOSIS :

INVESTIGATIONS :

Preliminary : Blood Group

Hb	Blood urea	ECG
TC	Ser. Creatinine	Echo
DC	Serum Na +	Urine – Alb
		– Sug
RBS		

Special :

X –ray Skull	- AP / Lat	
X – ray Orbit	- AP / Lat	
X – ray Chest	- PA / Lat	
X-ray optic foramen (Rheese view)		
CT Scan Brain	Plain / Contrast	
MRI		
CSF Analysis		
Hormonal Assay	-	T3 / T4 / TSH
	-	Serum estradiol
Angiography (cerebral)		
FFA		
EEG		

MANAGEMENT

Medical

Surgical
- Total Excision
- Subtotal excision & Biopsy

Radiotherapy

HISTOPATHOLOGY REPORT :

POST – OP / POST – Rx VISUAL PARAMETERS : Improved / Status quo / Deteriorated

FOLLOW UP :

MASTER CHART

MASTER CHART

S.No	Name	Age	Sex	MRD No	General Complaints				Neurological evaluation Abnormality of					Ocular Complaints				OPHTHALMIC EVALUATION												CT or MRI	HPE	Treatment
					H(± V)	Weakness (OT/Paralysis	Convulsions	Others	Other CN	Motor system	Sensory System	Gait & incoordination		Decreased Vn	Diplopia	Ptosis	Others	EOM		Corneal Sensation		Pupil		Fundus		V/A		Color Vn		Fields				
																		R	L	R	L	R	L	R	L	R	L	R	L	R	L			
1	PITCHAYEE	58	F	31043	-	-	-	R- Tinnitus Deafness	R VIII N	-	-	-		-	-	-	-	N	N	N	N	A	A	N	N	6/6	6/6	N	N	N	N	R CPA Mass	Acoustic Neuroma	VPSHUNT
2	CHITRA	25	F	10141	+	-	-	-	-	-	-	-		+	-	-	-	N	N	N	N	SA	SA	POA	POA	6/60	CFCF	N	NP	Temp HP	NP	Sellarmass with Suprasellar Extn	Pituitary Macroadenoma	R-FT Craniotomy excision
3	CHINNATHAMBI	43	M	10134	+	-	-	L- Deafness Giddiness	L XIII N	-	-	-		-	-	-	Nys	N	N	N	N	A	A	P	P	6/6	6/6	N	N	BSE	BSE	L CPA Mass	Acoustic neuroma	VPShunt + Surgery
4	THANGADURAI	29	M	10144	+	-	-	-	-	-	-	-		+	-	-	-	N	N	N	N	RAPD	A	POA	POA	PL	6/12	NP	Abn	NP	Temp HP	R Suprasellar Mass with Calcification	Cranio Pharyngioma	Surgery + RT
5	PERIASAMY	40	M	13117	+	-	-	LOC giddiness	-	Semi conscious		NP		-	-	-	-	VI Abn	VI Abn	N	N	A	A	P	P	NP	NP	NP	NP	NP	NP	FHyperdense Mass with calcification	Falx Meningioma	Craniotomy Excision
6	KUBENDRAN	12	M	6584	+	-	-	polydipsia polyuria	-	-	-	-		+	-	-	-	N	N	N	N	SA	SA	POA	POA	1/60	1/60	N	N	BTH		Sellar and Suprasellar Mass	Cranioph aryngioma	Surgery
7	RAMAKRISHNAN	37	M	14141	+	-	+	Vertigo, Tremors	-	-	-	+		-	-	-	-	N	N	N	N	A	A	N	N	6/6	6/6	N	N	N	Sup Qp	R. T-P Mass	Astrocytoma	R-Temp Craniotomy Subtotal excision
8	SAMEELA	21	F	25041	-	-	+	-	-	-	-	-		-	-	-	-	N	N	N	N	A	A	P	P	6/12	6/24	N	N	BSE	N	R. T-P Sol	Glioma	R-Temp Craniotomy Subtotal excision
9	VELUSAMY	64	M	25951	+	-	-	Deviation of angle of mouth, drooling of saliva	L- VII UMN	-	-	+		-	-	-	-	N	N	N	N	A	A	P	P	6/18	6/9	N	N	N	N	R. P-O Sol		
10	KRISHNA MOORTHI	40	M	21257	+	+	+	-	-	-	-	-		+	+	-	-	N	N	N	N	A	A	N	N	6/12p	6/6	N	N	N	N	R. Parasagittal sol (?Glioma)		
11	BANNARI	50	M	29032	+	-	-	LOC giddiness	-	-	-	-		-	-	-	-	N	N	N	N	A	A	P	P	6/24	6/36	N	N	N	N	R PARIETAL SOL		
12	ESWARAN	35	M	21935	+	-	-	L- Deafness	L-VIII	-	-	-		-	-	-	Nys	N	N	N	ABN	A	A	P	P	2\60	8\60	N	N	N	N	L CP angle sol	Schwannoma	Posterior Occipital Craniotomy And subtotal Excision
13	SUBRAMANIAM	52	M	26369	+	-	-	Unsteadiness during walking	-	+	-	-		-	-	-	LE PKP DONE	UPGAZE PALSY		N	N	A	A	N	N	6/12	6/36	N	N	N	N	R Occipital Sol With Obstructive Hydrocephalus	-	VP SHUNT
14	PADMA	37	F	35410	+	-	-	R-Deafness	R-VIII	-	-	-		-	-	-	Nys	N	N	ABN	N	A	A	P	P	6/12	6/12	N	N	N	N	R CP angle mass	-	-
15	PREMA	40	F	40970	-	-	-	-	-	-	-	-		+	-	-	-	N	N	N	N	A	A	POA	POA	3/60	3/60	N	N	BTH		Sellar Mass with Suprasellar Extension	Pituitary Macro Adenoma	Surgical Excision
16	KARTHIKEYAN	4	M	51286	+	+	-	R Hemi Paresis	-	+	-	+		-	-	-	-	N	N	N	N	A	A	P	P	NP	NP	NP	NP	NP	NP	Mass lesion R Thalamus And Midbrain	-	-
17	ABDULHAKEEM	39	M	31541	+	+	+	L Hemi Paresis	-	+	-	+		-	-	-	-	N	N	N	N	A	A	N	N	6/12	6/12	N	N	N	Sup Qp	R Temp. Sol	Glioma	Temp Cranio tomy
18	RANJIT KUMAR	10	M	49615	+	+	-	-	-	+	-	+		+	-	-	-	N	N	N	N	A	SA	PALE DISC	POA	6/60	NOPL	N	NP	TUB VN	NP	Suprasellar mass with Calcification	Cranio Pharyngioma	Surgery
19	PALANIAMMAL	30	F	229221	+	-	-	Numbness R Side of Face, Deviation of Angle of Mouth	R VII	-	-	-		+	-	+	-	III, IV, VI ABN	N	N	N	A	A	P	P	6/12	6/6	N	N	N	N	R Temp Sol	Meningioma	Surgery

MASTER CHART (contd...)

S.No	Name	Age	Sex	MRD No	General Complaints				Neurological evaluation Abnormality of				Ocular Complaints				OPHTHALMIC EVALUATION												CT or MRI	HPE	Treatment
					H(+V)	Hemiparesis /Hemiplegia	Convulsions	Others	Other CN	Motor/gait	Sensory System	Gait & coordination	Decreased VA	Diplopia	Ptosis	Others	EOM		Corneal Sensation		Pupil		Fundus		V/A		Color Vn		Fields				
																	R	L	R	L	R	L	R	L	R	L	R	L	R	L			
20	SHANTHA	65	F	25584	+	+	-	R- HEMIPA RESIS	R VII N	+	-	+	-	-	-	R EXPOSURE KERATITIS	N	N	N	N	A	A	P	P	6/60	6/36	N	N	N	N	L T-P SOL	GLIOMA	L PARIETAL CRANIOTOMY EXCISION
21	VELUMANI	57	F	31493	+	-	-	Giddiness	-	-	-	+	-	-	-	-	N	N	N	N	A	A	P	P	6/24	6/18	N	N	BSE	BSE	R CEREBELLAR SOL	-	VP-SHUNT
22	PREMA	14	F	31620	+	+	-	R- Hemiparesis	R VII N	+	-	-	-	-	-	-	VI ABN	N	N	N	A	A	P	P	6/12	6/9	N	N	N	N	L Parietal sol	Glioblastoma GR IV	VP Shunt Tumor biopsy, RT
23	MANONMANI	19	F	32178	+	-	-	-	-	-	-	-	+	-	-	-	N	N	N	N	SA	SA	POA	PAE DISC	1/60	6/36	N	N	N	N	Dorsal Arachnoid Cyst With Dilated Ventricles	-	-
24	CHINNASAMY	53	M	37141	-	-	-	R DEAFNESS	R VIII N	-	-	-	+	+	-	-	VI Abn	N	ABN	N	A	A	P	P	6/24	6/24	N	N	N	N	R CP Angle Sol	Acoustic Neuroma	VP Shunt
25	UDAYAKUMAR	10	M	10497	-	-	+	-	-	-	-	-	-	-	-	-	N	N	N	N	A	A	P	P	4/60	6/60	N	N	N	N	R Parietal sol with perilesional edema	-	-
26	NOORULLAH	41	M	34062	+	-	+	-	-	-	-	-	-	-	-	-	N	N	N	N	A	A	P	P	6/24	6/18	N	N	R HOM	HP	L TP SOL	GBM	-
27	GOVINDAMMAL	50	F	9945	+	-	-	L TINNITUS, DEAFNESS	L VIIIN, VIIIN	-	-	+	-	+	-	NYS	N	VI ABN	N	N	A	A	P	P	6/18	6/12	N	N	BSE	BSE	L CP Angle Sol	Hemanglo Blastoma	VP Shunt
28	SUBBULAXMI	55	F	35142	+	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	A	A	P	P	6/18	6/18	N	N	N	N	Mass lesion Posterior III Ventricle	-	VP Shunt
29	PALANISAMY	38	M	32413	+	-	+	-	-	-	-	-	-	+	-	-	VI ABN	VI ABN	N	N	A	A	P	P	6/12	6/9	N	N	L HOM	HP	R Temp Sol	GBM	R FT Craniotomy excision
30	MUSTAFA	45	M	35429	-	-	+	-	-	-	-	-	-	-	-	-	N	N	N	ABN	A	A	P	P	6/9	6/18	N	N	N	N	Multiple Meningiomas RFP and LCPA	-	R FP Craniotomy excision
31	SARASWATHI	42	F	36215	+	-	+	-	-	-	-	-	+	-	-	TOV	-	-	N	N	A	A	P	P	6/36	6/60	N	N	N	N	B/L Multiple Contr. Enhancing Lesions. Frontal & Cerebellum	-	-
32	BAKIALAKSHMI	48	F	37803	+	-	+	Altered behaviour	-	-	-	-	+	-	-	-	N	N	N	N	RAPD	A	Pale Disc	N	6/60	6/12	N	N	N	N	R. Frontal sol	Astrocytoma Grade II	R F CRANIOTOMY EXCISION
33	THULASIMANI	40	F	37007	+	-	-	L- Deafness	L VIII N	-	-	-	-	-	-	-	N	N	N	ABN	A	A	P	P	6/12	6/12	N	N	N	N	L CP Angle Sol	Acoustic Neuroma	VP SHUNT
34	VIJAYA	35	F	15431	+	-	-	Amenorhoea Wt gain, Change in Voice	-	-	-	-	+	-	-	-	N	N	N	A	RA PD	PALE DISC	POA	6/12	3/60	N	ABN	Sup QP	Temp HP	Sellar mass with SS and optic canal extension	Pituitary Adenoma	SURGERY
35	RAVICHANDRAN	37	M	32455	+	-	-	-	-	-	-	-	+	-	-	Loss of Field of Vision	N	N	N	N	A	RA PD	Temp Pallor	POA	6/9	NO PL	ABN	NP	Temp HP	NP	Suprasellar Sol	Cranio pharyngioma	SURGERY
36	EMELI	12	F	14123	-	-	+	-	-	-	-	-	+	-	-	-	N	N	N	N	A	A	PPOA	PPOA	1/60	CF CF	NP	NP	NP	NP	R Frontal cystic lesion with calcification	-	-
37	SAROJINI	52	F	12156	-	-	-	-	-	-	-	-	-	-	+	"	N	III,IV ABN	N	N	A	6mm NA	N	N	6/9	6/12	N	N	N	N	Meningioma L Cavernous Sinus	-	-
38	SAGAYAMARY	67	F	12218	+	-	-	-	-	-	-	-	-	-	-	-	N	III,IV ABN	N	N	A	A	N	N	6/9	6/12	N	N	N	N	L Parasellar Cavernous sinus sol	Meningioma	-
39	VALLIAMMAL	47	F	41017	+	-	-	Numbness R.Side of Face	-	-	-	-	+	-	+	"	III N	N	ABN	N	8mm NA	N	POA	N	CFCF	6/6	NP	N	NP	N	R Petrous sol	Meningioma	SURGERY
40	KALPANA DEVI	2	F	41314	+	-	-	LOC	"	-	-	-	+	-	-	"	NP	NP	N	N	SA	SA	POA	POA	NP	NP	NP	NP	NP	NP	Postr fossa sol with obstructive hydrocephalus	-	-
41	AROKIYASAMY	58	M	42140	+	-	-	-	-	-	-	-	-	-	-	Proptosis LE	N	N	N	N	A	A	N	N	N	N	N	N	N	N	L high parietal sol with bony erosion	Meningioma	SURGERY
42	RAMAN	32	M	41310	-	-	+	Previous craniotomy scar	-	-	-	-	+	-	-	"	N	N	N	N	SA	SA	POA	POA	6/60	5/60	N	N	BTH		R Suprasellar sol	Recurrent Anaplastic Oligodendro-glioma	RT

MASTER CHART (contd...)

S.No	Name	Age	Sex	MRD No	General Complaints				Neurological evaluation Abnormality of				Ocular Complaints				OPHTHALMIC EVALUATION												CT or MRI	HPE	Treatment
					H(+V)	Hemiparesis /Hemiplegia	Convulsions	Others	Other CN	Motor/gait	Sensory System	Gait & coordination	Decreased VA	Diplopia	Ptosis	Others	EOM		Corneal Sensation		Pupil		Fundus		V/A		Color Vn		Fields				
																	R	L	R	L	R	L	R	L	R	L	R	L	R	L			
43	MANJULA	10	F	40610	+	-	+	-	-	-	-	-	-	-	-	-	N	N	N	N	A	A	P	P	6/12	6/9	N	N	N	N	L Cerebellar sol	Astrocytoma	Subocciptal craniotomy excision postop RT
44	IBRAHIM	51	M	49018	+	+	-	L Hemiparesis	L VII N	+	-	+	-	-	-	-	N	N	N	N	A	A	P	P	6/12	6/12	N	N	N	N	R FP SOL	GBM	-
45	PRASHANTH	15	M	51954	+	-	-	-	L VII N	-	-	-	-	+	-	Nys	N	VI ABN	N	N	A	A	P	P	6/6	6/6	N	N	N	N	Calcified Posterior Fossa sol with Obstructive Hydrocephalus	Ependy-moma	Surgery
46	SUBERA	9	F	14231	+	-	-	-	-	-	-	-	+	-	-	-	N	N	N	N	RAPD	A	POA	Temp Pallor	PL	4/60	NP	N	NP	Tub Vn	Sellar Mass with Suprasellar Extension	Cranio Pharyngioma	Surgery
47	CHINNAMMAL	60	F	16132	+	-	-	Enlargement of hands feet	-	-	-	-	+	-	-	-	N	N	N	N	RAPD	A	POA	N	HM	6/12	NP	N	NP	Temp HP	Sellar Mass with Parasellar Extension	Pituitary Adenoma	Surgery
48	PAPPATHI	14	F	17394	+	-	-	LOC	-	-	-	+	-	-	-	-	N	N	N	N	A	A	P	P	6/18	6/18	N	N	N	N	Midline Posterior Fossa sol	Cerebellar Astrocytoma	Surgery + RT
49	GANAPPATHI AMMAL	48	F	25766	+	-	+	-	-	-	-	-	+	-	-	-	N	N	N	N	SA	SA	PPOA	PPOA	PL	PL	NP	NP	NP	NP	Frontal lobe sol with mass effect on both optic nerves	Meningioma	-
50	MUTHUKUMAR	19	M	24131	-	-	-	"	-	-	-	-	+	-	-	-	N	N	N	N	RAPD	A	N	N	HM	6/6	NP	ABN	NP	Temp HP	Suprasellar Mass with Calcification	Cranioph aryngioma	Surgery
51	LASAHOURRIA	19	M	15431	+	-	-	-	-	-	-	-	+	+	-	-	VI ABN	VI ABN	N	N	A	A	P, MF	P, MF	6/60	2/60	N	N	Tub vision	Tub vision	R TP sol	GLIOMA	R T-P Craniotomy excision RT

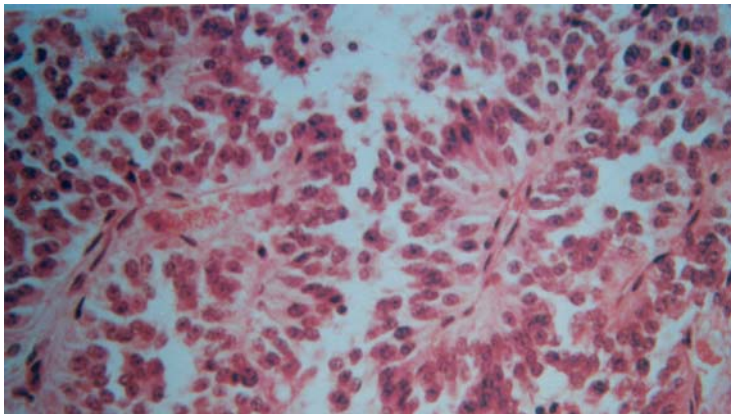
ABBREVIATIONS

ABBREVIATIONS

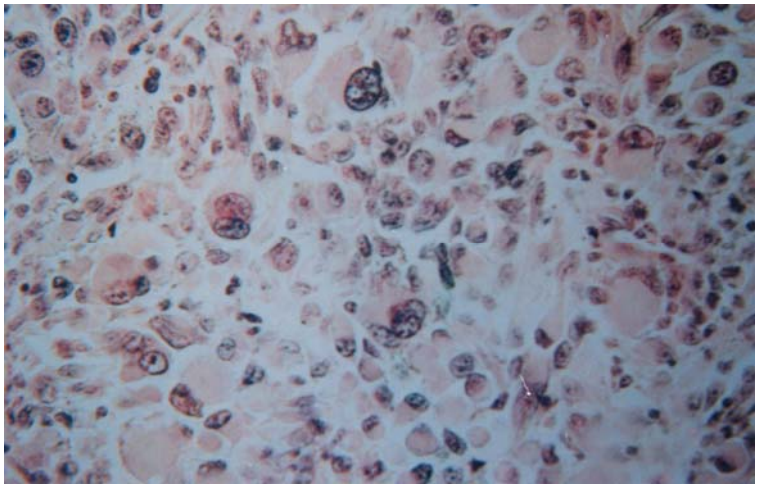
A	-	Acting
Abn	-	Abnormal
B/L	-	Bilateral
B&B	-	Bowel & Bladder
BSE	-	Blind Spot Enlargement
BTH	-	Bitemporal hemianopia
CFCF	-	Counting Fingers Close to face
CN	-	Cranial nerves
CPA	-	Cerebello Pontine angle
EOM	-	Extra – ocular movements
F	-	Frontal
F-T-P	-	Fronto – Temporo – Parietal
F T	-	Fronto Temporal
H	-	Headache
HM	-	Hand movements
Hom	-	Homonymous
Hp	-	Hemianopia
HPE	-	Histopathological examination
Inf.	-	Inferior
L	-	Left
LMN	-	Lower Motor Neuron palsy
LOC	-	Loss of Consciousness
ME	-	Macular edema
N	-	Normal
NA	-	Not acting
Nas.	-	Nasal
NP	-	Not Possible

Nys	-	Nystagmus
P	-	Papilledema
PL	-	Perception of light
POA	-	Primary Opic atrophy
Qp	-	Quadrantanopia
R	-	Right
RAPD	-	Relative afferent pupillary defect
RT	-	Radiotherapy
SA	-	Sluggishly acting
SOL	-	Space Occupying Lesion
SS	-	Suprasellar
Sup.	-	Superior
T-P	-	Temporo – Parietal
Temp.	-	Temporal
TOV	-	Transient Obscurations of Vision
UMN	-	Upper Motor Neuron palsy
V/A	-	Visual Acuity
V	-	Vomiting
V-P	-	Ventriculo – Peritoneal
Vn	-	Vision
↓	-	Decreased
↑	-	Increased
-	-	Absent
+	-	Present

CASE NO. 19. MENINGIOMA



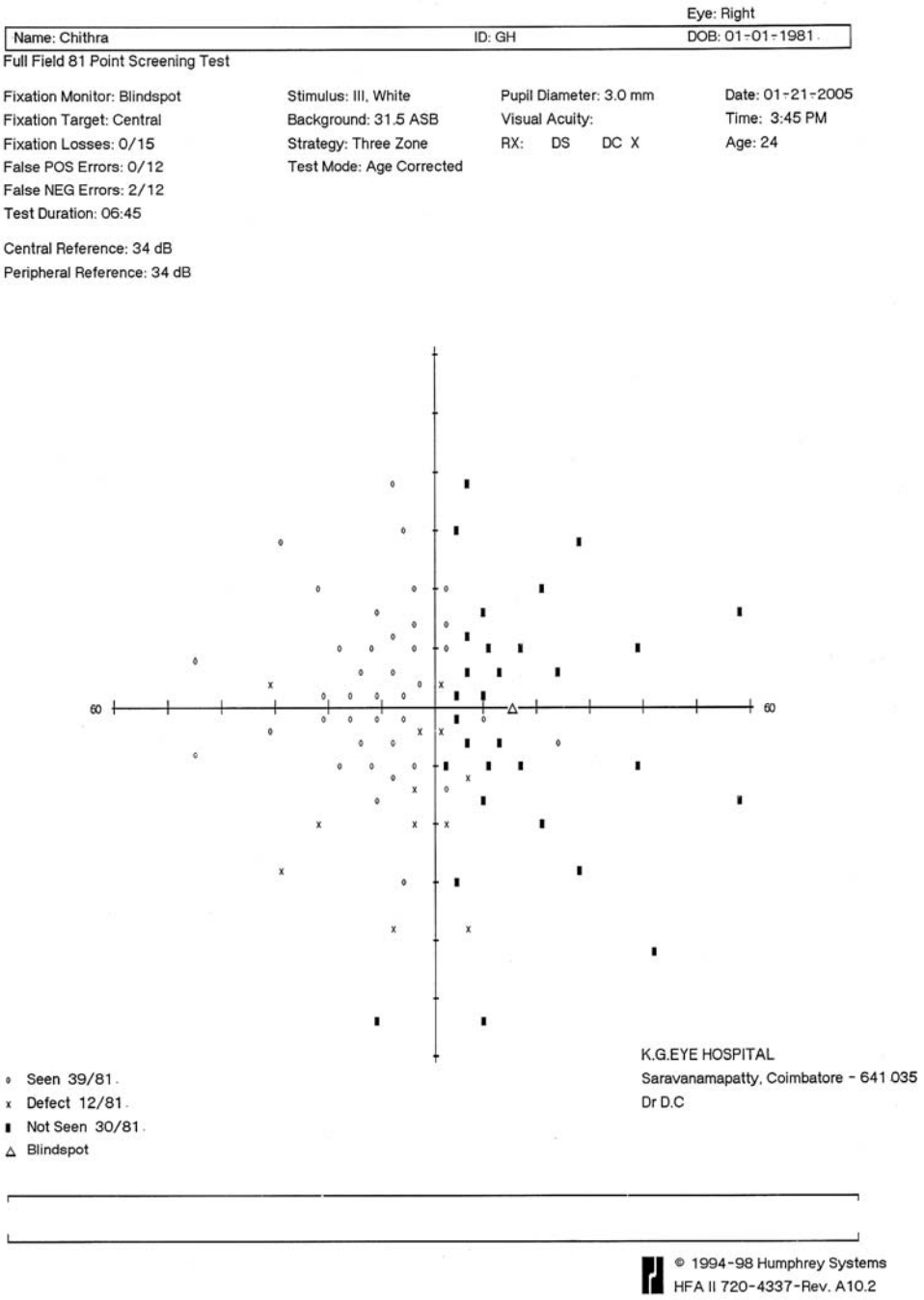
CASE NO. 20. GLIOMA



CASE NO. 2. PITUITARY ADENOMA

SHOWING

TEMPORAL HEMIANOPIA (RE)

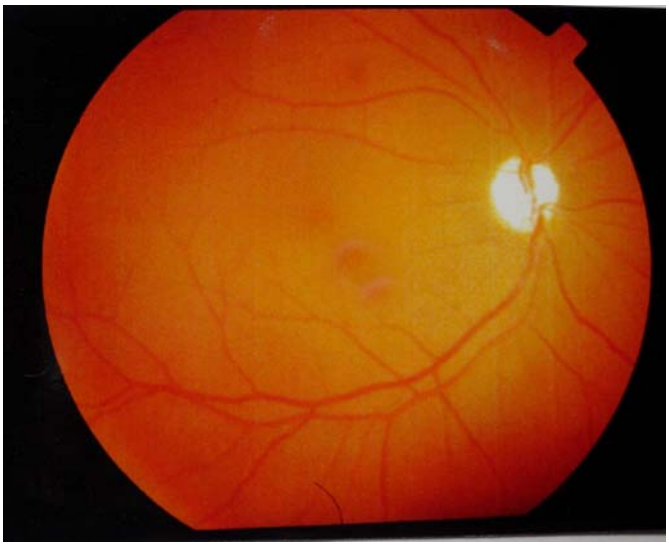


PAPILLEDEMA

Courtesy : Clinical Ophthalmology. Jack J Kanski 5th edn.

FLUORESCEIN LEAKAGE IN PAPILLEDEMA

CASE NO. 46 . PRIMARY OPTIC ATROPHY

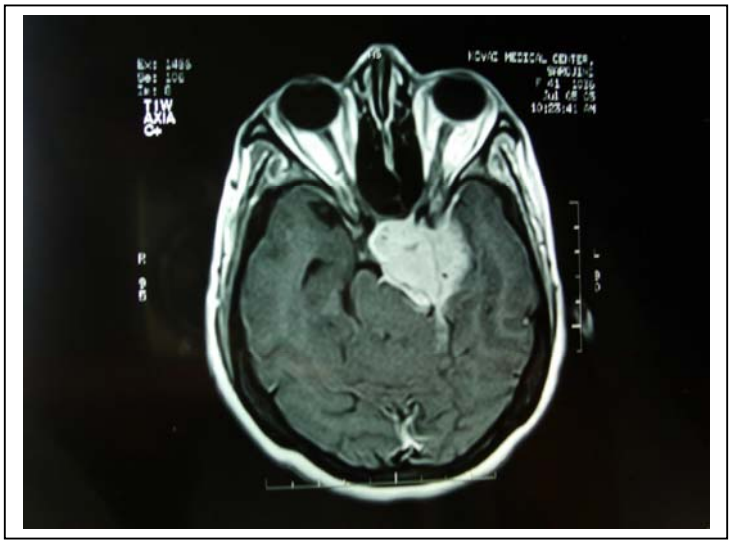


CASE NO. 38 . SHOWING COMPLETE PTOSIS (LE)

CASE NO. 37 . LEFT III CN PALSY

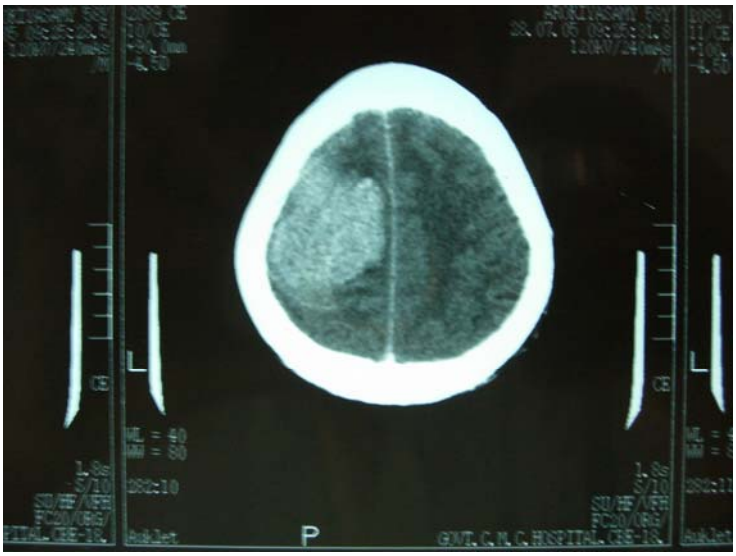
CASE NO. 37 . LEFT VI CN PALSY

CASE NO. 37. MRI SHOWING MENINGIOMA LEFT CAVERNOUS SINUS

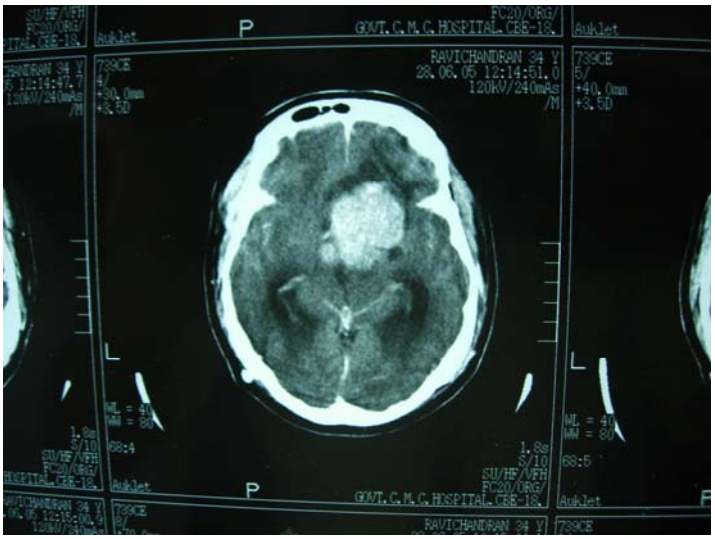


CASE NO. 30. CT SHOWING RIGHT FRONTOPARIETAL MENINGIOMA

CASE NO. 41. CT SHOWING LEFT HIGH PARIETAL SOL

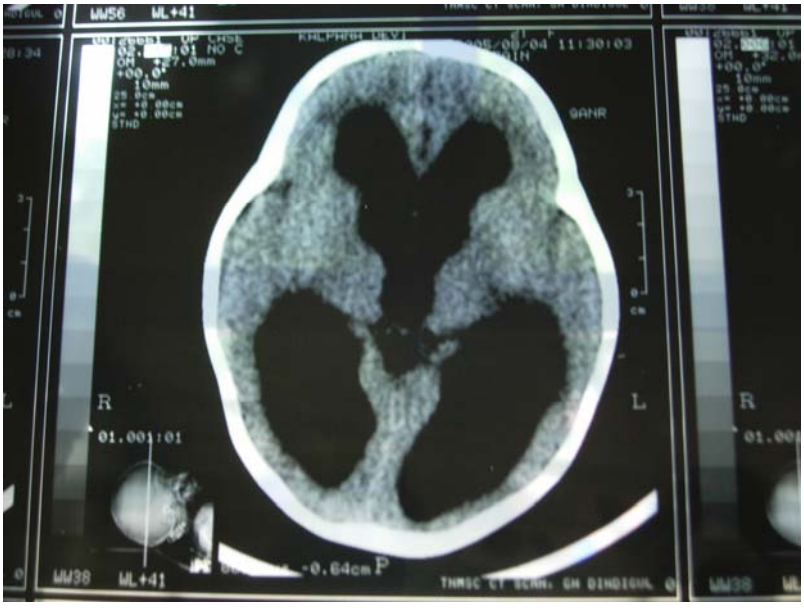


CASE NO. 35. CT SHOWING SUPRASELLAR CRANIOPHARYNGIOMA

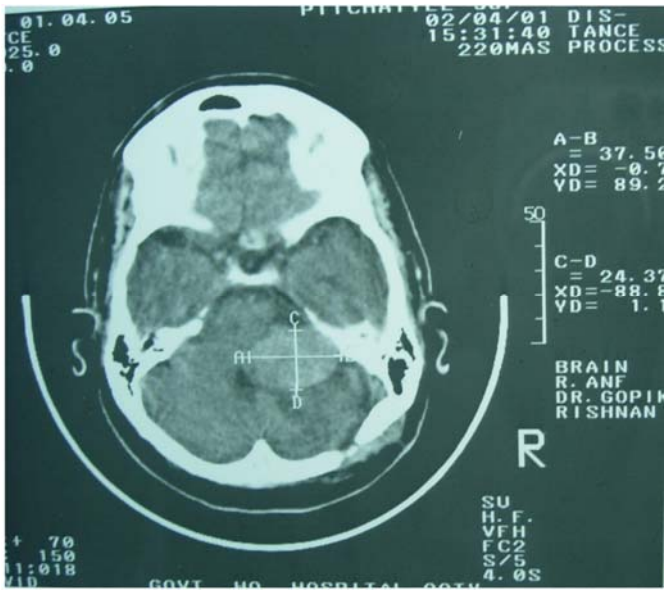


CASE NO. 39. SHOWING RIGHT III CN PALSY

CASE NO. 40. CT SHOWING OBSTRUCTIVE HYDROCEPHALUS WITH DILATED VENTRICLES.



CASE NO. 1. CT SHOWING RIGHT CPA TUMOR



CASE NO. 20. ESTABLISHED PAPILLEDEMA